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EYE, EAR, NOSE AND THROAT

A MANUAL FOR STUDENTS AND PRACTITIONERS

BY

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NEW SECOND EDITION, THOROUGHLY REVISED

ILLUSTRATED WITH 180 ENGRAVINGS AND 8 COLORED PLATES

LEA & FEBIGER
PHILADELPHIA AND NEW YORK
1917

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PREFACE TO THE SECOND EDITION.

THE constant demand for a work that presents briefly, clearly, and reliably affections of the eye, ear, nose and throat has stimulated the authors to rewrite almost every chapter of this volume. Such important modifications of and additions to our knowledge of these subjects have been made that a less radical revision would have been as unsatisfactory to themselves as to their readers.

To facilitate reference, the anatomy of the parts has been introduced and two colored plates have been added. The various methods of examining the ear, including the functional tests, have been especially elaborated in order that the student may be more readily led to a logical diagnosis. Diseases of the middle ear have been presented in the order of their occurrence which it is hoped may simplify their study. A brief chapter upon Malformation of the External Nose has been inserted, and the authors' method of utilizing the Sluder technic for tonsillectomy, together with other modern methods, is fully described and illustrated. The prescriptions have been subjected to careful revision, and are retained.

The authors are indebted to Dr. George McBean for many valuable suggestions and for reading the proofs of the section relating to the ear, nose, and throat.

As in the previous edition, Dr. Wippern is responsible for the section upon the Eye and its Affections, and Dr. Ballenger for the remainder of the book.

H. C. B.
A. G. W.

CHICAGO, 1917.

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THE EYE.

EXAMINATION OF PATIENTS.

GENERAL CONSIDERATIONS.

THE examination of a patient with eye trouble should be preceded by the history of the case. Traumatism, exposure to cold, foreign bodies, disturbance of vision, injudicious use of a poultice, and the coexistence of general diseases are often points of value in making a diagnosis.

Inspection.—Inspection of the individual will at times suggest the possible cause of the ocular trouble. Jaundice, pregnancy, or chronic alcoholism might explain poor vision at night. A waxy color of the skin and edema of the lids might suggest Bright's disease; which in its turn would explain the disturbance of vision due to a retinitis; as would also the existence of pregnancy. The gait of a tabetic person would suggest a cause of optic atrophy. Inability to close an eye (lagophthalmos), next to exposure to cold, is due to ear disease involving the facial nerve. Ptosis with a paralytic squint in young adults generally means syphilis. The lesions of *acquired syphilis* explain the existence of paralysis, retinitis, and iritis. The evidence of *hereditary syphilis*, as notched teeth, scars at the angles of the mouth, and peculiar shape of the cranium, explain the cause of corneal trouble. The presence of a large quantity of mucopurulent secretion in the conjunctival sac signifies conjunctival disease. Photophobia occurring in children most often implies corneal disease. The broad face, concave nose, and large nostrils suggest the possibility of atrophic nasal disease and the reason for stricture of the nasal duct. A small, thick nose with an eczema of the introitus is often accompanied by conjunctival or corneal disease. Anemia, scrofula, rheumatism, diabetes, gout, and similar diseases that depress the vital powers, may cause ocular affections which should be regarded simply as expressions of such general disease.

The diseases of the eye may be either *organic*, in which case there is a lesion; or *functional*, when simply an error of refraction, as hypermetropia, myopia, or astigmatism, is present.

The organic affections of the eye and appendages which can be diagnosed by simple inspection yield to treatment, as a rule, better than those which require the use of an ophthalmoscope, since they are more accessible for the application of treatment. Hence diseases of the lids, cornea, conjunctiva, iris, and lens are more amenable to treatment than affections of the choroid, retina, and optic nerve.

The correction of *refractive errors* constitutes a large part of eye work, and is very satisfactory; but in order that this be properly done it is often necessary to understand the significance of pathological changes which interfere with the refraction and perception of light.

INSTRUMENTS.

An *ophthalmoscope*, a *biconvex lens* of about 16 D., known as an object-lens, and a complete *trial-case* with test-types and astigmatic chart are absolutely indispensable for the examination of the eyes. A *retinoscope*, *perimeter*, and *ophthalmometer* are very valuable adjuncts. A host of other instruments have been devised, most of which are superfluous.

The Ophthalmoscope.—An ophthalmoscope (Fig. 1) is an instrument containing a more or less concave perforated mirror and a number of convex and concave lenses of different strengths, so arranged that they may be brought, preferably one at a time, behind the aperture of the mirror. Perhaps the most complete and best constructed ophthalmoscope is that made after the pattern of Morton (Fig. 1). The arrangement of the lenses and the presence of two concave and one plane mirror make it a superior instrument. A much cheaper instrument, after the Loring pattern (Fig. 2), is, however, just as good for ordinary cases, and is even preferred by some. A *biconvex lens* of 16 D. is almost a part of an ophthalmoscope.

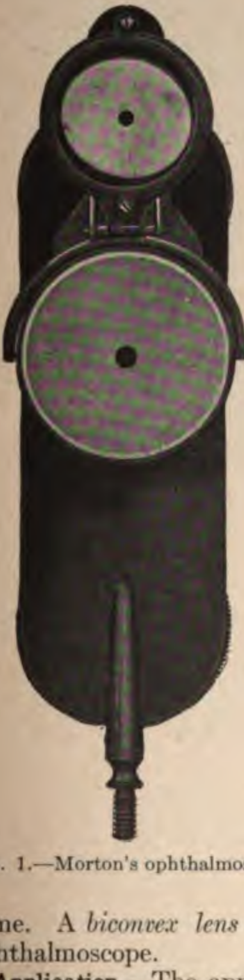


FIG. 1.—Morton's ophthalmoscope.

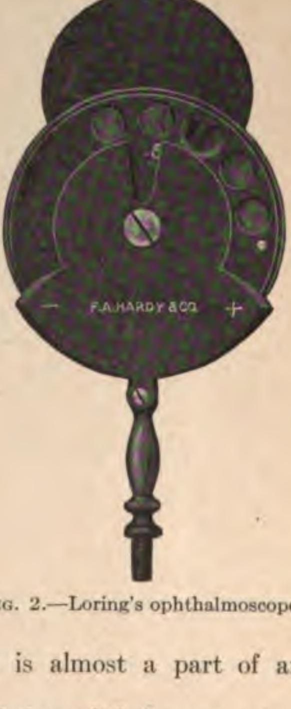


FIG. 2.—Loring's ophthalmoscope.

Application.—The application of the ophthalmoscope is a wide one. Not only can intra-ocular diseases be recognized and the location of opacities be determined, but the refractive condition of the eye can also be estimated. The pathological changes observed in the eye often assist in diagnosticating cerebral, renal, and general disease. The recognition of most intra-ocular pathological conditions is not very difficult, but the determination of refractive errors will be found very unsatisfactory unless the examining person has had great experience and is in practice. Fortunately, glasses need not be prescribed after an ophthalmoscopic examination, now that retinoscopy can so satisfactorily be applied; retinoscopy is an objective test and requires little skill.

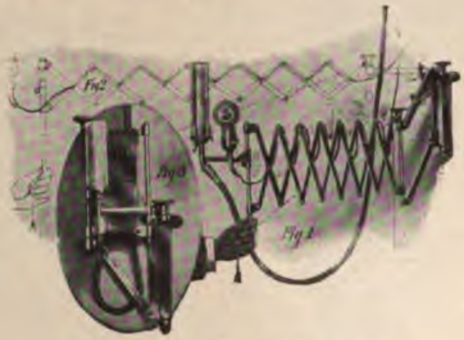


FIG. 3.—Illuminating apparatus.

Ophthalmoscopic examination should take place in a darkened room, and the source of light (Fig. 3) may be an Argand burner in the absence of electricity, but an electric lamp with a frosted bulb which prevents the image of the filaments in the eye is preferable.

The student usually experiences some difficulty in acquiring the skill necessary to use the ophthalmoscope, and as it is hardly fair to patients to subject them to tedious, prolonged and only too often useless examinations by beginners, a schematic eye (Fig. 4) should be employed until the technic has been mastered. Various fundus conditions may be studied in the schematic eye so that the student not only acquires the technic but also learns to interpret the diseased conditions of the fundus.

Method of Examination.—The method of ophthalmoscopic examination is either direct or indirect.

Direct Method.—To examine a patient by the direct method, the light should be on a level with the patient's ear and on the side of the eye to be examined. The observer reflects the light into the patient's eye, using his right eye and standing or sitting at the patient's right side to examine the patient's right eye; and using his left eye and standing or sitting at the left of the patient to examine the left eye. The mirror of the ophthalmoscope should be as close as possible to the patient's eye.

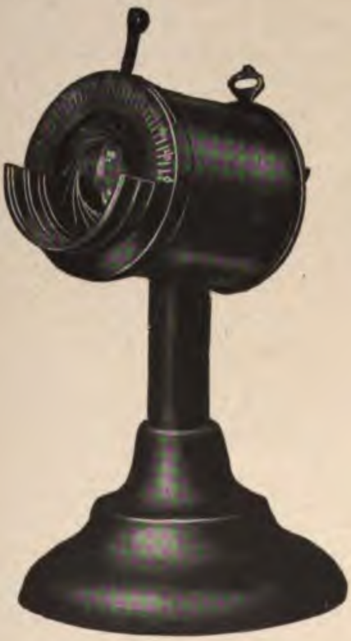


FIG. 4.—Schematic eye.

The *image* obtained by the direct method is *erect*, and is magnified about 16 times, regardless of the distance between the patient's and the observer's eye.

Indirect Method.—To examine a patient by the indirect method the light and position should be the same as in the direct, but a biconvex lens of 16 D. should be placed at about 2 inches in front of the eye to be examined, the ophthalmoscope being about 18 inches away. This method gives an *inverted image* of the fundus and magnifies the image four times if a biconvex lens of 16 D. be used.

The ophthalmoscopic examination should be preceded by one with the aid of *oblique* or *focal illumination;* the biconvex lens of 16 D. is so held that the rays of light from the lamp, which should be at the side and slightly in front of the patient, are condensed upon the part to be examined.

To examine the *cornea* the glass should be held farther from the eye than when an examination of the *lens* is undertaken. By an examination with oblique illuminations, opacities of the cornea and the anterior portion of the lens, the relative depth of the anterior chamber, iridodonesis, and changes of the iris can be easily recognized. Changes in the peripheral and posterior parts of the lens, and even in the anterior portion of the vitreous, can be observed when the pupil is dilated. An examination by oblique illumination will explain a disturbance of vision from opacities otherwise overlooked, and will save in such cases much time and trouble.

The ophthalmoscopic examination of an eye can often be satisfactorily made without the use of a mydriatic; but in some cases, more numerous with beginners, dilatation of the pupil will be found necessary. A few instillations of a 4 per cent. cocaine solution will suffice to dilate the pupil unless there be adhesions between the iris and lens or cornea.

To examine the *refracting media* the ophthalmoscope should be held about 8 inches from the eye examined, the observer being emmetropic. For a myope the distance should be less; and a hyperope or presbyope would require a lens of + 3 or + 4 D. or more in the aperture. An opacity cannot only be seen, but, by observing the parallactic displacement of the opacity when fixed with reference to the margin of the pupil, its location determined.

An *opacity in the cornea* seems to move in a direction opposite to the movement of the mirror; one upon or near the anterior capsule remains stationary in all positions of the mirror; and one near the posterior capsule seems to move in the same direction.

In order to examine the cornea or lens *very carefully* a convex lens of 16 to 20 D. should be brought into the aperture. After dilating the pupil the crystalline lens should be examined with a + lens of 16 D. and not even an incipient cataract will escape detection.

The refraction of the eye may be determined with the ophthalmoscope by either the direct or indirect method, but the former is almost exclusively employed.

When both the observer's and the patient's eye are emmetropic, the ciliary muscle being relaxed, the details of the fundus can be distinctly seen through the aperture.

When the patient is myopic and the observer emmetropic, the concave lens necessary to show the details of the fundus represents the degree of myopia.

In the same way the degree of hypermetropia is determined by the strength of the convex lens necessary to show the fundus distinctly.

When the observer is either hypermetropic or myopic, the same rule applies; but the observer must have corrected his refractive error.

If the observer will notice the *movement of the vessels,* whether it is with or against the movement of the mirror, ametropia can be inferred: when there is hypermetropia the vessels move "with;" when myopia, "against."

Astigmatism is demonstrated in an eye by the direct method (Fig. 5), when the optic disk is seen elongated in the direction of the meridian of greatest refraction. The retinal vessels are most distinctly seen in the meridian of the greatest refractive power. It goes without saying that the apparent elongation of the disk will coincide with the astigmatic meridan in myopia and will be at right angles to it in hypermetropia.

When the *indirect method* (Fig. 6) is employed, the eye being emmetropic and the accommodation relaxed, the image of the disk remains the same in size and shape for every distance of the biconvex lens from the eye. With an increase of this distance in hypermetropia the image becomes smaller; and in myopia, larger; when no astigmatism is present, the original shape is always retained. When astigmatism is present, recession of the lens causes variation in both size and shape of the image of the disk. The elongation of the disk occurs at right angles to the meridian of greatest refraction, provided the lens is at a distance from the eye less than its focal distance. At its focal distance from the eye the disk would appear circular; and beyond that distance would seem elongated at right angles to the long axis of the image when the lens is within its focal distance from the eye.

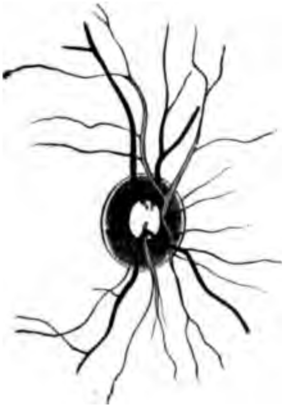


Fig. 5.—Erect image of disk in astigmatism, with meridian of greatest refraction nearly vertical. (de Wecker and Jäger.)

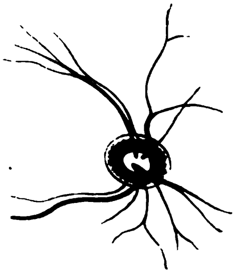


Fig. 6.—The same disk as shown in Fig. 5, seen by the indirect method. (de Wecker and Jäger.)

The direct and indirect methods may be employed to determine *differences of level in the fundus;* but the former method is almost exclusively employed. A difference of refraction of two points in the fundus amounting to 3 D. means $\frac{1}{25}$ inch difference of level. In case of papillitis with a swollen disk or glaucoma with cupping, this method may be employed to measure respectively the amount of swelling or cupping.

Trial-case. Test-types. Astigmatic Chart.—The trial-case, test-types, and astigmatic chart (Figs. 7, 8, 9), like the ophthalmoscope, are indispensable, and for the correction of refractive errors constitute the best resource. With a complete trial-case supplied with prisms, the relative strength of the extra-ocular muscles and the degree of the heterophorias, and even of strabismus, can be ascertained.

The test-types, including a chart of illiterate distance-type and one of test-type for near tests, are of value to determine the visual acuity, with or without glasses. In order to test the acuity, a space 20 feet in length should be available, since at that distance the rays which come to a focus upon the retina are parallel and the accommodation is not required. The visual acuity of most cases should be determined and recorded in order to discover disturbance of vision. The *test-types* are arranged in rows which, according to the size of the letters, can be read by a normal eye at specified distances. When the row marked 20 can be read at 20 feet, the vision is written $\frac{20}{20}$, and is normal. When the row marked 120 is the smallest that can be seen at 20, the feet vision is written $\frac{20}{120}$. When all the letters but three can be read in the row marked 40 at 20 feet, the vision is expressed $\frac{20}{40}-3$. The *illiterate distance type* is of value in the examination of both children and adults who cannot read the Roman letters. Such patients who have sufficient intelligence can with their

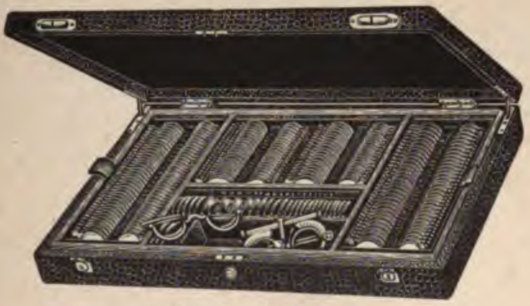


Fig. 7.—Trial-case.

children or foreigners, should (after ophthalmoscopic examination) be subjected to a retinoscopic examination and fitted accordingly.

A complete trial-case should contain the following:

30 pairs each of convex and concave spherical lenses from 0.25 D. to 20 D.

20 pairs each of convex and concave cylindrical lenses from 0.25 D. to 6 D.

16 prisms, from ½ degree to 20 degrees.
1 plano blue and 1 plano red glass.
6 plano smoked glasses, 6 shades.
1 solid disk of rubber and 1 ground-glass disk.
1 pin-hole disk.
1 stereoptic disk.
1 Maddox rod.
1 Maddox double prism (improved).
1 graduated trial-frame.
1 plain trial-frame.

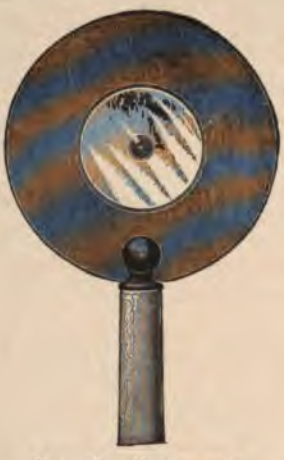


Fig. 10.—Retinoscope.

The Retinoscope.—The retinoscope (Fig. 10) is the most useful instrument for the objective determination of refractive errors. It is nothing more than a plane or concave mirror perforated at the center and supplied with a handle. The Thorington retinoscope has a plane mirror and can be recommended.

Retinoscopy is the method of determining the refraction by observing the change of illumination of the pupil that occurs when the mirror of the retinoscope is rotated on its vertical and horizontal axes. When the mirror is turned on its vertical axis, the refraction of the horizontal meridian is determined; and when on its horizontal axis, the refraction of the vertical meridian may be computed. In order to *practise retinoscopy* a dark room and a single source of light, as an Argand burner, are necessary. The *light* may be placed directly over the patient's head; or, when Thorington's method is employed, the burner, which should be covered with a chimney in which there is a round aperture of about one-half inch diameter, is placed near the head of the examining person, who reflects the light from the aperture to the patient's eye. The *distance* between the patient and the examined person may vary, 20, 40, or 80 inches being the distance employed to suit the case. When the *mirror is turned* on its vertical axis, it will be noticed that the edge of the shadow is usually vertical, and that the shadow moves horizontally across the pupil, either in the direction of the movement of the mirror or against it. In the former case the shadow is said to move "with," in the latter, "against."

When a *plane mirror* is used, the distance of the mirror from the examined eye being about 40 inches, the rules are as follows:

The mirror rotated ⟶:

Shadow "against" implies myopia more than 1 D.

A series of concave lenses should be placed before the examined eye, the other being covered, beginning with a weak one. The last concave lens which causes the shadow to go "against," increased by 1 D., is the refraction.

When the shadow moves quickly, it indicates a low degree of myopia; when slowly, a high degree.

Shadow "with" implies myopia less than 1 D., emmetropia, or hypermetropia.

A series of convex lenses should be placed before the examined eye, the other being covered, beginning with a weak one. The first convex glass which causes the shadow to move "against," diminished by 1 D., is the refraction.

When the shadow moves quickly, it indicates emmetropia, myopia less than 1 D., or a low degree of hypermetropia.

When a *concave mirror* is used, the rules for the movements of the shadows given above are reversed. When the distance between the mirror and the examined eye is 20 inches, then 2 D. are either subtracted or added to the finding, as the case may be.

When the distance is 80 inches, or 2 meters, 0.5 D. is either added or subtracted.

In an *emmetropic*, or normal, eye the shadow will be reversed by placing before it a convex lens of 1 D., if the distance between the mirror and the examined eye is the focal distance of a glass of 1 D.—that is, 40 inches. Hence the rays of light emerging from the cornea were parallel before the lens was used.

Suppose it be found that a + 3 D. is the weakest convex lens necessary to cause reversal of the shadow (the distance between the mirror and the eye being 40 inches) in both the vertical and horizontal meridians, then the condition may be formulated thus: $\overset{+3}{+}\;+3$. After subtracting 1 D., the refractive condition would be expressed thus: $\overset{+2}{+}\;+2$. Since a convex lens of 2 D. causes normal refraction in both meridians, a convex spherical lens satisfies the condition.

Suppose a + 3 D. reverses the shadow in the vertical meridian and a + 4 D. is necessary in the horizontal meridian, then the refractive condition of the eye may be expressed thus: $\overset{+2}{+}\;+3$. The correcting glass is a convex spherical lens of 2 D. (which corrects the vertical meridian and 2 D. of the horizontal) combined with a cylinder of 1 D. (the difference between 3 and 2), its axis being at right angles to the meridian to be corrected—hence vertical or at 90 degrees. The glass may then be expressed + 2 sph. ⊂ + 1 cyl. × 90 or + 2 sph. ⊂ + 1 cyl. axis vertical. This case is one of compound hypermetropic astigmatism.

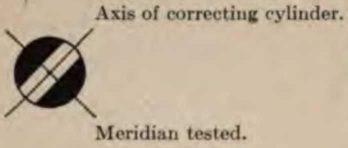
In a case of *simple myopia* of 3 D. it will be found that a

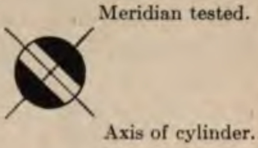
– 2 D. is the strongest concave glass which does not reverse the shadow in both meridians. When a – 4.5 D. is the strongest concave glass which does not reverse the shadow in the vertical meridian, and a – 3 D. the strongest glass for the horizontal meridian, the refraction would be expressed: $\overset{-5.50}{\text{———}}-4$. The correction would be – 4 sph. ⊃ – 1.5 cyl. × 180 for this case of compound myopic astigmatism.

Suppose a – 3 D. is the strongest concave glass that does not reverse the shadow in the vertical meridian, and a + 3 D. is the weakest convex glass that reverses the shadow in the horizontal meridian, then the refraction would be expressed thus: $\overset{-4}{\text{———}}+2$. This is a case of mixed astigmatism. The correcting glass might be designated in three ways: – 4 cyl. × 180 and + 2 cyl. × 90; – 4 sph. ⊃ + 6 cyl. × 90; or + 2 sph. ⊃ – 6 cyl. × 180. The last-mentioned way is preferred by the optician, who likes working – cylinders onto + spheres.

In converting two cylinders into a sphere and cylinder this fact should be kept in mind: a sphere is equal to two cylinders of the same kind and degree when the axes are at right angles to each other: $+2 \text{ sph.} = \begin{cases} +2 \text{ cyl.} \times 180 \\ +2 \text{ cyl.} \times 90 \end{cases}$ Take the example above: – 4 cyl. × 180 combined with + 2 cyl. × 90. By using a + 2 sph. in place of a + 2 cyl. × 90, the glass is too strong by a + 2 cyl. × 180. Therefore it is necessary to subtract a + 2 cyl. × 180, which is the same as adding a – 2 cyl. × 180 to – 4 cyl. × 180, which makes a – 6 cyl. × 180.

When the shadow *moves horizontally* from side to side, the edge of the shadow is vertical, but the horizontal meridian is the one tested. A cylinder to correct an error must be placed at right angles to the meridian requiring correction. Since the edge of the shadow is at right angles to the meridian tested, the position of the edge of the shadow represents the axis of the cylinder required for correction. Hence by noting the position of the edge of the shadow the axis of the correcting cylinder is determined:





Occasionally the axis of the correcting cylinder will be other than horizontal or vertical, and the retinoscopic examination will often determine the proper axis.

The student who has a schematic eye, such as is recommended to acquire the art of ophthalmoscopy, will find it of very great use to perfect himself in retinoscopy. By adjustment and use of lenses every possible refractive error may be produced, and a little practice will give the student the facility necessary to compute the refractive condition in the patient's eye.

The Perimeter.—The perimeter (Fig. 11) is an instrument devised to determine the field of vision.

The field of vision can be determined in a general way by the hand, and better yet by a graduated blackboard; but the use of a perimeter is the most accurate and scientific method.

To determine the field of vision with the *hand*, the patient should stand with his back to the window and the examiner should have his eyes on a level with those of the patient. When the right field is to be determined, the patient whose left eye is covered should be directed to look continually at the examiner's left eye. The examiner then moves his left hand in various positions, and in a relative way the size of the field of vision can be determined.

When a *blackboard* is used, this should be divided into meridians and concentric circles by lines, and the patient is directed to fix his eye at the centre, the other eye being covered. The chalk is then advanced on the blackboard at the greatest distance within the field of view, and this line gives the field of vision. The various parts of the retina are measured at different distances by this method, which is for this reason inferior to perimetric examination.

The *perimeter* (Fig. 11) is a band of metal which is divided into degrees, beginning at its middle. It turns upon an axis at the middle point in such a way that it passes through all meridians. A slide is moved along the arc so that concentric circles can be made within 90 degrees. In order to determine the field for various colors, a white, blue, red or green button may be used. This is inserted into the slide.

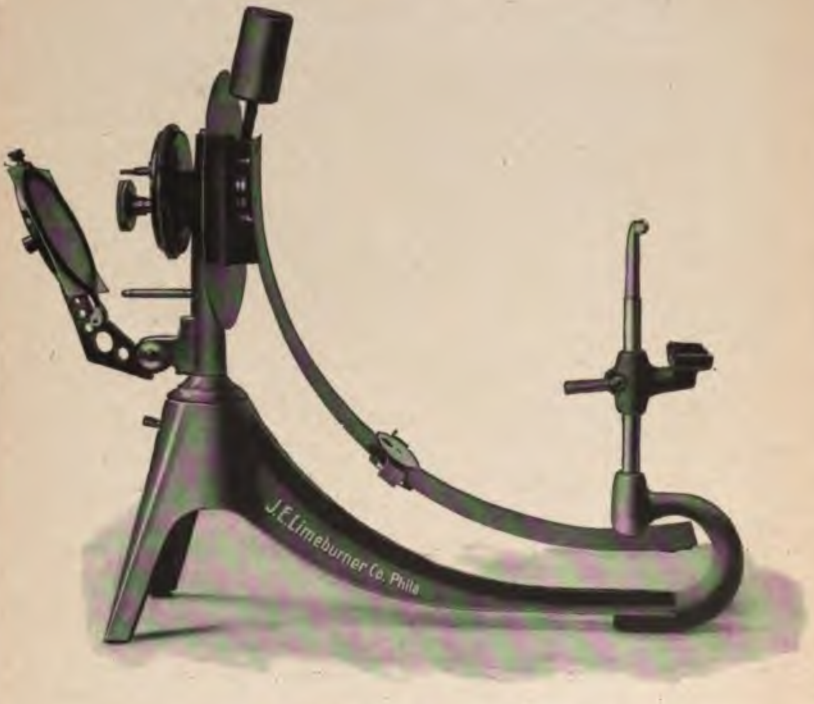


Fig. 11.—McHardy's self-registering perimeter.

The *patient* is seated with his chin in an adjustable rest in such a position that the examined eye is directly in front of the middle of the arc and the other eye is covered. A card made specially for the right or the left eye is placed at the back of the axis of the arc and turns with the arc. The card is divided into meridians and concentric circles, and is so placed that movements of the arc correspond with the meridians on the card. The point nearest the periphery at which the patient can see the slide on a given meridian is noted on the card, and these points are ascertained for all the meridians. A line connecting these points determines the visual field (Fig. 12). Circumscribed areas which cannot be seen in the visual field are called *scotomata*.

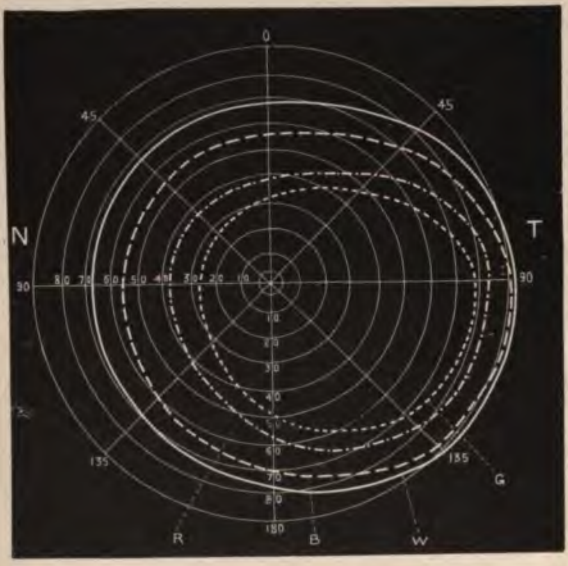


FIG. 12.—Field of vision of right eye as projected by the patient on the inner surface of a hemisphere, the pole of which forms the object of regard (half-diagrammatic). *T*, temporal; *N*, nasal side; *W*, boundary for white; *B*, for blue; *R*, for red; *G*, for green. (Landolt.)

Scotoma.—A scotoma may be central, peripheral, or annular, and may exist for only certain colors. Normally the fields of vision for various colors are not the same, and the examination of the visual field to determine the relative visual fields for colors is of value in the diagnosis of disease. A diminution of blue means disease of the retina; of red and green, the optic nerve. Overlapping of the color fields is frequently indicative of hysteria.

Blind Spot.—In making a perimetric examination it will be observed that a blind spot is always present to the temporal side of the point of fixation between 10 and 20 degrees. This is due to the optic disk, which is not sensitive.

Hemiopia.—Hemiopia, which implies that one lateral half only of the retina is functioning, can be easily demonstrated with the perimeter. When the left halves of the visual field of both eyes are intact, the hemiopia is said to be *homonymous*. The same applies to the right side. *Temporal hemiopia* implies that only the temporal side of the retina of both eyes is functioning, and hence the visual field is intact on the nasal sides. Homonymous hemiopia occurs from a lesion in either the right or left optic tract. Temporal hemiopia is the result of a lesion near the middle of the optic chiasm.

Concentric Contraction.—When the visual field is narrowed at the periphery so that the limits of the visual field are equally diminished the condition is called concentric contraction. This condition is often observed in retinitis pigmentosa and occasionally in glaucoma.

Scotomata occur frequently in choroidal or retinal disease when the lesion is circumscribed.

The Ophthalmometer.—The ophthalmometer (Figs. 13, 14) is an instrument for measuring the corneal curvature of the eye, and for determining the amount and character of corneal astigmatism by noting the variations in that curvature. It consists mainly of two bright or luminous objects (mires) whose images are reflected by the corneal surface; of a telescope through which the images are seen enlarged; and a birefringent prism within the tube of the telescope, which brings the images into apparent proximity. Each mire has a long central line which is in exact alignment with the corresponding line on the other mire; and a shorter line, or spur, at right angles to the larger one, which may be brought to appear in alignment with its fellow on the other mire by moving the prism within the tube. The prism is provided with a rack, and is moved forward or backward in the tube by means of a pinion which carries a graduated scale.

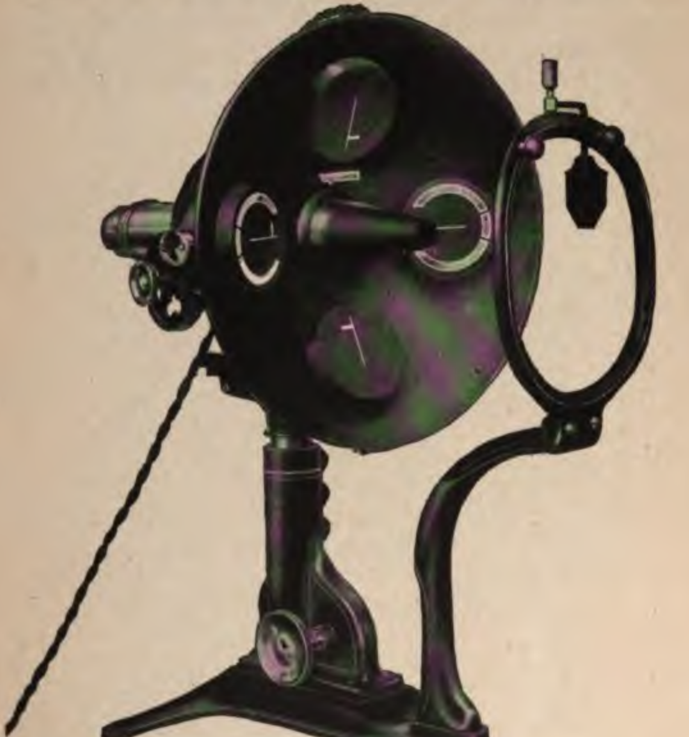


FIG. 13.—The C.-I. ophthalmometer (front view). This is a front view showing the mires, the head rest, the eye cover, the vertical adjustment and the prism adjustment wheel from which we may read the radius of curvature and the refracting value of each of the principal meridians.

Each cornea examined, acting as a graduated reflector, shows between the spurs of the images an amount of space inversely proportional to its diminishing power—that is, its curvature. The prism is moved nearer the eye or farther from it until this distance between the spurs is exactly eliminated. The scale attached to the pinion will then indicate the curvature in diopters. The entire apparatus as described may be rotated before the eye to be examined and the curvature measured at various meridians. The longer lines of the mires will appear in exact alignment upon an astigmatic cornea only when they are in the meridians of greatest or least curvature—that is, at the axes. The difference between the curvature at the two axes is the amount of corneal astigmatism.

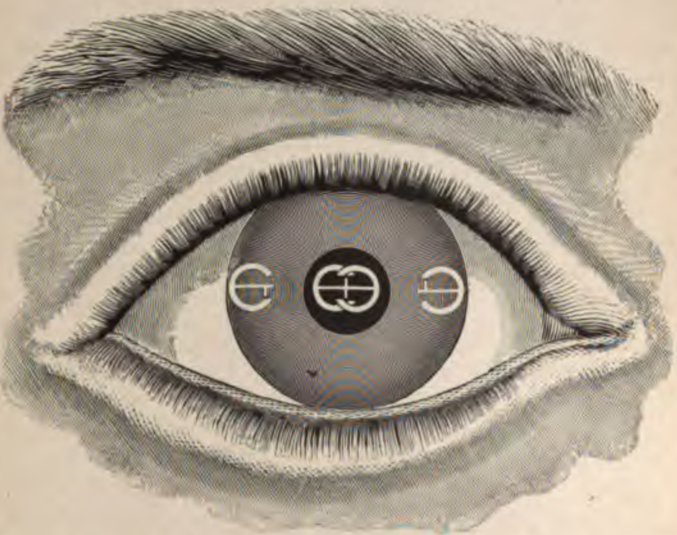


FIG. 14.—Measuring the radius. When the eye piece is in the primary position, and the meridian lines form an unbroken line, the mires are in what is known as the primary position. The radius of curvature in this position may be measured by rotating the adjusting wheel on the right side of the telescope until the spurs on the meridian lines are seen opposite each other, or until a perfect cross is formed.

It is evident that the ophthalmometer affords a rapid and objective method to determine the amount and axis of corneal astigmatism, and that its intelligent use will often save time and trouble in correcting errors of refraction.

THE ORBIT.

ANATOMY OF THE ORBIT.

The orbits (Plate I) are two cavities which contain the organs of vision. Seven bones, the frontal, sphenoid, ethmoid, superior maxillary, malar, lacrimal, and palate, enter into the formation of each orbit. The bony walls of the orbits have their periosteum, and next to this, mesially, is more or less fatty cellular tissue, which forms a bed of support for the globe. Next comes the capsule of Tenon, which is "serous" membrane consisting of a visceral and a parietal layer. The parietal layer clothes the cavity of the fatty tissue in which the globe is imbedded; and the visceral layer covers the posterior part of the globe from near the margin of the cornea to the entrance of the optic nerve. The ocular muscles near their insertion pierce the capsule of Tenon which gives off some fibers to reinforce the sheaths of the muscles. The orbit contains periosteum, fatty cellular tissue, the lacrimal gland, the capsule of Tenon, the ocular muscles, nerves, bloodvessels, and bulb.

MALFORMATIONS OF THE ORBIT.

In acephalous monsters the anterior opening of the orbits is directed upward; in double-faced monsters with three or four eyes the orbits are not fully developed; and in cases of cyclopia the single common orbital cavity is of small size and the optic foramina are at times wanting. In cases of microphthalmos and anophthalmos the orbit is in part defective. When loss of the eyeball occurs in infancy, the orbital cavity does not always attain the mature size. In such a case a rubber ball or wax cast should, if possible, be worn to supplant the bulb, in order to encourage the development of the orbit.

NEOPLASMS OF THE ORBIT.

Tumors of the orbit may arise in the bony walls and periosteum, the optic nerve, or in the cellular tissue. They produce exophthalmos, strabismus, and more or less loss of motility.

The bony walls of the orbit may be the seat of osteomas. These are usually very hard, and their removal is often possible only after enucleation of the eyeball. The danger from meningitis and cerebral abscess is great in removal of osteomas of the roof of the orbit.

The cellular tissue of the orbit may be the seat of cystoid and dermoid tumors, angiomas, lipomas, and sarcomas.

The early removal should be thoroughly performed in all cases of orbital tumors.

INJURIES OF THE ORBIT.

The margin of the orbit, because of its prominence, is subject to injury from blows or falls.

Wounds.—*Contusions* and *incised* or *lacerated wounds* often occur in the soft parts overlying the margin of the orbit, and usually cause little trouble.

Fracture of the malar bone may cause anesthesia from injury of the infra-orbital nerve and diplopia from injury of the inferior oblique muscle. Some injuries involve both the supra-orbital and infra-orbital nerves.

Fractures of the Orbital Walls.—Fractures of the orbital walls produce such *hemorrhage* that there are usually exophthalmos and ecchymosis of the lids and conjunctiva. When a fracture of the orbital wall extends to the optic foramen the sight may be lost.

Gunshot Wounds.—Gunshot wounds may produce immediate blindness from injury to the optic nerve or rupture of the choroid, although the eyeball appears uninjured.

When the lacrimal bone can be easily displaced from the orbital plate of the ethmoid and when hemorrhage from the nose occurs, emphysema of the orbital tissues being present or not, there is a fracture of the *inner orbital wall.*

Emphysema.—Emphysema of the orbital tissues may be produced by fractures, causing a communication between the orbit and the antrum of Highmore, the frontal or sphenoidal sinuses, the ethmoidal cells, or the nasal duct.

Injuries, muscle operations, and foreign bodies may cause orbital abscesses. These should be opened by exploratory punctures, and relief will be given even though no pus be found.

Fractures of the orbital walls should be *treated* according to the rules of surgery. Detached pieces of bone must be removed; and should an abscess develop, this is to be thoroughly drained. Emphysema of the orbital tissue usually terminates favorably with avoidance of sneezing and coughing and application of a light bandage.

DISEASES OF THE ORBIT.

Orbital Cellulitis.—Orbital cellulitis is an acute, subacute, or chronic inflammation of the fatty cellular tissue, and may be idiopathic or traumatic in origin.

When *acute*, the disease presents a formidable appearance. The pain in the orbit is excruciating. There are fever, headache, edema of the lids, chemosis of the ocular conjunctivæ, exophthalmos, neuroretinitis, ulceration and suppuration of the cornea, and panophthalmitis. When due to erysipelas, the most severe symptoms are likely to develop. Thrombosis of the orbital veins and cavernous sinus may occur in the course of an orbital cellulitis.

Treatment.—The treatment depends upon the severity of the symptoms. Numerous incisions through the conjunctiva, or through the lids if it be impossible to open them, should be made to relieve the tension and secure drainage. Irrigation of the wound with bichlorid of mercury solution (1 to 2000) should be made from time to time. Hot applications hasten the process. If complicated by abscess of the cornea and panophthalmitis, evisceration of the bulb may be required. Enucleation is not so safe as evisceration in these cases, since cerebral complications are more likely to occur after the former.

Periostitis.—Periostitis is due to injury, cold, foreign bodies, or syphilis.

When *acute*, there are slight fever, great pain, tenderness on pressure, and the lids are red and swollen. Caries or necrosis of the underlying bone may result after a time, which can be ascertained by probing the fistulous opening which usually occurs through the lids.

In *treating* this condition the fistula should be enlarged if necessary, and the dead bone removed. The wound should be kept clean and open to favor healthy closure from the bottom. Acute periostitis should be treated as an orbital cellulitis by puncture and drainage.

When *due to syphilis*, the pain is greater at night. For these cases antisyphilitic treatment should be instituted.

Foreign bodies causing periostitis should be removed.

Tenonitis.—Tenonitis, or *inflammation of the capsule of Tenon*, may be of idiopathic or traumatic origin.

The upper lid is swollen and edematous, and movement of the eyeball is painful. Exophthalmos, when present, is slight.

The *idiopathic* form is often due to rheumatism or gout.

Hot fomentations to the lids and antirheumatics and anodynes usually bring about recovery.

In the *traumatic* form, antiseptic solutions should be used to cleanse the conjunctival sac; and free incisions should be made as soon as the formation of pus is evident.

Exophthalmic Goiter.—Basedow's or Graves's disease causes more or less exophthalmos and draws attention to the eyes. Enlargement of the thyroid gland, tachycardia, and protrusion of the eyeball are the cardinal symptoms of the disease, which is primarily an affection of the nervous system.

When the cornea suffers from inability to close the lids (lagophthalmos), it may be necessary to use a bandage or perform a tarsorrhaphy, whereby the lids are united only to such an extent as not to interfere with vision.

Pulsating Exophthalmos.—Pulsating exophthalmos presents the following symptoms: protrusion of the eyeball, noises audible over the orbit, and pulsation felt on the eyeball and about the orbit. It may be spontaneous or traumatic. Most of these cases are due to rupture of the internal carotid artery in the cavernous sinus.

Treatment.—The treatment is compression of the common carotid artery; and if done without result, ligation is indicated.

THE EYELIDS.

ANATOMY.

The eyelids (Fig. 15) cover and protect the eyeball.

The palpebral fissure is the space between the free margins of the lids; the outer angle is known as the *outer canthus*, the inner angle as the *inner canthus*.

The **upper lid** is larger than the lower, and has a special muscle to raise it, the *levator palpebra superioris*. The free margin of the lid has an outer and inner lip. The *outer lip* is rounded and contains two or three rows of hairs, known as the *cilia*. The inner or posterior lip contains the mouths of the *Meibomian glands;* and near the inner canthus the *lacrimal papilla*, at the summit of which is the *punctum lacrimale*.

From without inward the lids are composed of integument, subcutaneous areolar tissue, muscles, fibrous tissue (known as *tarsal cartilage*), Meibomian glands, and conjunctiva.

The **integument,** which is studded with fine, downy hairs, is very thin, and because of the loose connection with the areolar tissue is lax.

The **subcutaneous areolar** tissue (containing no fat) connects the integument and muscle. It contains **hair follicles** and **sweat glands.**

The following **muscles** are in the lids: the palpebral portion of the orbicularis palpebrarum, the ciliary muscle of Riolanus, the tensor tarsi, the aponeurosis of the levator palpebræ superioris, and the superior and inferior palpebral muscles.

The **orbicularis palpebrarum** is a sphincter muscle, and consists of two portions: an orbicular, acting for the most part involuntarily; and a palpebral, subject to the will. When both portions act together, the lids are brought into close contact, as occurs in photophobia. The palpebral portion is concerned in gently closing the eyes. It is supplied by the facial nerve.

The *muscles of Riolanus* is that small part of the orbicularis palpebrarum between the Meibomian glands and the follicles of the cilia.

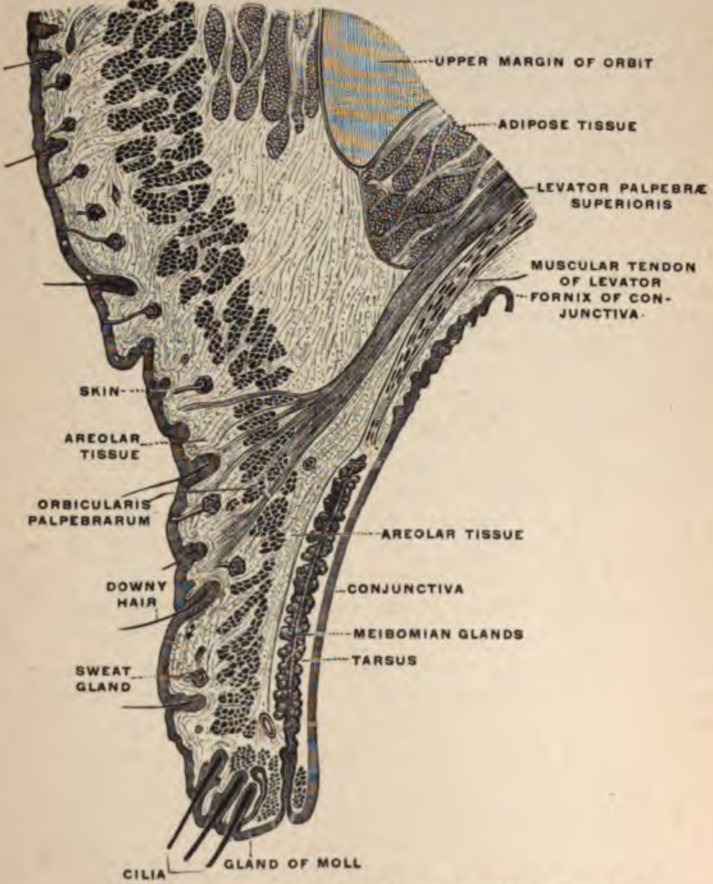


Fig. 15.—Upper lid in sagittal section. (After Merkel.)

The *tensor tarsi*, or *Horner's muscle*, is that portion of the orbicularis palpebrarum situated at the inner side of the orbit arising from the crest of the lacrimal bone and adjoining surface of the orbit. It draws the eyelid inward, and by compressing the punctum lacrimale on the globe facilitates the drainage of tears.

The **levator palpebræ superioris** is the antagonist of the orbicularis palpebrarum, for it raises the eyelid. It arises near the upper margin of the optic foramen, and is inserted by a broad aponeurosis into the anterior surface of the tarsal cartilage. It is supplied by the third nerve.

The **superior palpebral** is a small involuntary muscle composed of non-striated muscle fibers. It arises from between the fibers of the levator palpebræ superioris just before it becomes aponeurotic, and is inserted into the under surface of the upper margin of the tarsal cartilage.

The **inferior palpebral** muscle arises from the orbital connective tissue. They are supplied by the sympathetic.

The **tarsal cartilages** are two plates of dense fibrous connective tissue. The superior is of semilunar form measuring about one-half inch at its broadest part and tapering toward the extremities. The *lower* smaller and thinner tarsal cartilage is elliptical in form. It measures one-quarter inch at its broadest part. The outer and inner extremities of each cartilage are held to the margin of the orbit by the outer and inner *canthal ligaments*. The inner canthal ligament is known as the *tendo-palpebrarum.*

The **Meibomian glands** are imbedded in grooves in the inner surface of the tarsal cartilages. There are from 30 to 35 in the upper lid and from 20 to 30 in the lower lid. They correspond in length with the breadth of the tarsal cartilages, and hence those of the upper lid are the longer. A Meibomian gland consists of a straight tube with a blind extremity, into which small secondary follicles open. These sebaceous glands supply a secretion which prevents adhesion of the lids. The infant has very large Meibomian glands.

The outer lip of the free margin of the lids has protruding from it two or three rows of hairs, the cilia. Those of the upper lid curve upward and are longer than those of the lower lid, which curve downward.

Small **sebaceous follicles** (the glands of Zeiss), and modified sweat glands (known as Moll's glands), are among the roots of the cilia which lie in the connective tissue between the orbicularis palpebrarum and the tarsal cartilage. One of Zeiss's glands opens into each hair follicle, and the secretion serves to lubricate the cilium. The mouths of Moll's glands open just behind the cilia.

MALFORMATIONS AND CONGENITAL ANOMALIES.

Coloboma.—Coloboma is a fissure of the lid; it may be congenital or traumatic. The upper lid is more often the seat of the congenital form. It is sometimes associated with dermoid growths of the cornea or with colobomas of the iris and choroid.

Treatment.—The treatment consists in freshening the edges and stitching them together.

Epicanthus.—Epicanthus is a malformation in which a crescentic fold of skin overlaps the inner canthus. This condition is often associated with ptosis and strabismus.

The overlapping skin may be excised; or the operation of cutting out an oval piece of skin from the bridge of the nose, the long axis being vertical, and closing the wound by sutures improves the condition.

Ectropion, entropion, ptosis, distichiasis, ablepharia (partial or total), **blepharophimosis, symblepharon, ankyloblepharon,** and **cryptophthalmos** may be congenital.

NEOPLASMS (Benign).

Xanthelasma.—Xanthelasma, or *xanthoma,* has been classified with neoplasms; but recent researches have shown that it is the product of the degeneration of embryonically misplaced muscle fibers.

Xanthelasma *occurs* as yellowish-white patches seldom raised above the skin. This condition is most often found after middle age, particularly in females. The patches generally occur on the upper lid, near the inner canthus.

Treatment.—The treatment, usually desired for cosmetic effect, is excision.

Molluscum Contagiosum.—Molluscum contagiosum is a small round tumor affecting the sebaceous glands. It may attain the size of a pea; the top is flattened, and in the centre is a dark spot, representing the aperture of a follicle. It is not contagious, as was once thought.

Treatment.—The treatment consists in evacuation of the cheesy mass; sometimes it is necessary to excise the walls of the cavity and apply a strong solution of silver nitrate.

Angioma.—A *nevus*, or "mother's mark," is always congenital, and may cover the whole lid. When small, it may be successfully *treated* by the application of caustics, such as nitric or chromic acid, or by the actual cautery.

Telangiectasis is a bright-red patch occurring in the skin and subcutaneous tissue, and consists of enlarged capillaries, arterioles, and venules.

Cavernous angioma is a vascular tumor composed of anastomosing sinuses with a framework of connective tissue.

These tumors are often encapsulated, and can then be enucleated. When extensive, electrolysis is the best treatment. Multiple punctures with the electrolytic needle are made around the base of the tumor.

Papillomas.—Papillomas (due to hypertrophy of the papillæ of the skin) should always be removed, since they are sometimes the starting-point of epitheliomas.

Fibromas, enchondromas, myxomas, lipomas, lymphangiomas, and **neurofibromas** but rarely occur on the lids.

NEOPLASMS (Malignant).

Epithelioma.—Epithelioma begins on the skin usually at the border of the lid, and unless excised early, invades the bulb and even the orbit. It occurs more frequently on the eyelids than elsewhere. It commences as a small inflamed elevation, whose surface becomes excoriated and secretes a watery or bloody liquid which forms a crust or scab. This falls off from time to time, showing an indurated ulcerated surface. The progress of the ulceration is very slow, and pain is not severe unless the deeper structures be involved.

When thoroughly removed at the beginning, epitheliomas may not recur, but after they are large and have ulcerated, an operation is not satisfactory. The use of the x-rays and radium have in some cases proved beneficial, and electric desiccation is said to have produced cures.

Rodent ulcer is a form of epithelioma which leaves cicatricial tissue in the part invaded. The ulcers of lupus vulgaris and secondary syphilis are liable to be confounded with epithelioma.

Epithelioma is a disease of advanced age; syphilis, of youth or middle age. Epithelioma is much slower in progress than syphilis. Lupus commences usually before puberty and is slower in progress than epithelioma.

Sarcoma.—Sarcoma of the eyelid is nearly always secondary. It is characterized by rapid growth and early recurrence. Thorough removal is the *treatment.*

INJURIES OF THE EYELIDS.

The eyelids are often the seat of *incised* or *lacerated wounds, contusions, burns,* and *scalds.*

Owing to the laxity of the skin, *edema* and *ecchymosis* of the lids may be considerable.

As the result of injury either to the eyelid itself or to the orbit, producing periostitis and caries, *abscess* of the lid is not uncommon. *Ulcers* sometimes occur, the result of scalds, burns, or contusions. A *traumatic coloboma* may be the result of a vertical wound where no union by first intention takes place.

Emphysema of the lids is a symptom of fracture of one or more bones entering into the formation of the orbit. The source of the air producing this condition is from the frontal or superior maxillary sinuses, the ethmoidal cells, or the nasal cavity. When by reason of sneezing, coughing, or blowing the nose the air in the spaces mentioned is under pressure, air is forced into the areolar tissue at the seat of fracture.

Edema may be due to congestion in the lids, the conjunctiva, or the orbit, and is often an expression of diseases of the heart and kidneys. When the swelling is excessive, puncture and application of pressure relieve the condition.

Ecchymosis is an extravasation of blood in the subcutaneous tissue, and the most common cause is contusion.

Treatment.—The best treatment immediately after the injury is the application of cold, either ice or iced water. In order to *promote absorption*, the application of heat and a firm bandage are recommended.

Abscess is usually the result of contusions, although spontaneous abscesses occasionally occur in scrofulous or poorly nourished children. The application of heat hastens the process and a horizontal incision is indicated to evacuate the pus.

Ulcers from whatever cause should be treated by attention to cleanliness and the application of antiseptics.

Since *emphysema* is caused largely by blowing the nose, the proper conditions being present, the avoidance of this act is the only treatment.

Wounds should be treated in accordance with the rules of surgery.

SKIN DISEASES.

The eyelids may be the seat of many skin diseases, but the consideration of a few of the more common or peculiar will suffice.

Eczema.—Eczema is the most common of the skin diseases. The etiology is often the same as in other parts, yet, owing to the irritation of the skin by the tears, either as the result of rubbing with the hands or conjunctivitis, lacrimal catarrh, or ectropion, the lower lid may become affected. Inflammation, itching, and moisture make the diagnosis easy.

Treatment.—The treatment varies evidently with the cause. For *simple cases* the application of Hebra's ointment (equal parts of lead plaster and vaseline), zinc salve, or a 5 to 10 per cent. solution of silver nitrate is the usual treatment.

When the eczema is *secondary* to other diseases, the necessary treatment should be applied to them.

Erysipelas.—Erysipelas of the eyelids occurs in the course of facial erysipelas, and the superficial form is without danger.

The *phlegmonous type* may involve the capsule of Tenon and the deeper tissues of the orbit, and impair or destroy the sight from optic neuritis or atrophy. A meningitis from extension of inflammation along the optic-nerve sheath is rare and is usually fatal.

Treatment.—The treatment should consist in local applications of ichthyol, guaiacol, or 10 per cent. solution of silver nitrate and a supporting medication. The success of the treatment will depend largely upon the strength of the patient.

Herpes Zoster Ophthalmicus.—Herpes zoster ophthalmicus is a neuropathic disease characterized by the formation of vesicles along the course of one or more branches of the ophthalmic nerve. Neuralgia and fever may precede the eruption, which, owing to the attending redness and swelling, simulates erysipelas.

The administration of anodynes to relieve the pain and the dusting of the affected parts with zinc oxide or rice flour are indicated.

Milium.—Milium is a term applied to concretions of sebaceous matter about the size of a millet-seed. Simple expression is the *treatment*. This disease may be mistaken for the commencing stage of xanthelasma palpebrarum, the distinguishing features of which are the clear yellow color, limitation to the eyelids, and the flat end, plate-like arrangement of the lesions.

Spontaneous Ulcers.—Spontaneous ulcers of the lid may be due to lupus, syphilis, or scrofula.

Treatment.—The treatment of the ulcer of lupus consists in the use of the curette or actual cautery and the application of *x*-rays. The syphilitic ulcer is usually a late manifestation of syphilis, and should be diagnosed early, since antisyphilitic treatment is effective, and surgical measures superfluous and even dangerous. The scrofulous ulcer demands constitutional as well as local treatment.

Inflammation along the ciliary margin, *blepharitis ciliaris*, appears in two forms: squamous and ulcerative.

Blepharitis.—Blepharitis Squamosa.—Blepharitis squamosa is a subacute or chronic disease of the border of the eyelid characterized by the formation of scales and exudation of a tenacious secretion, often gluing the cilia together. On removal of the scales or crusts a more or less hyperemic surface presents itself, and often several lashes are pulled out in the process. The lashes, however, usually grow again.

Blepharitis Ulcerosa.—Blepharitis ulcerosa differs from the squamous form in that the inflammation is deeper, the secretion purulent, and the loss of cilia permanent.

Etiology.—Anemia, scrofula, eruptive fevers, neglect of cleanliness, exposure to dust, heat, winds, and irritants produce bilateral disease. Corneal opacities, a high degree of hypermetropia (causing strain of accommodation), and uncorrected errors of refraction are exciting causes. Chronic catarrhal conjunctivitis, phlyctenular conjunctivitis, trachoma, and epiphora are local causes. When epiphora produces blepharitis, the cause should be ascertained, whether increased secretion or disease of the lacrimal canal or sac. Blepharitis may cause a chronic catarrhal conjunctivitis, a stye, loss of the cilia, tumefaction along the border of the lids (particularly the upper), epiphora from eversion or narrowing of the puncta, trichiasis, and ectropion.

Treatment—This depends upon the cause. When con-constitutional treatment is needed, the iodide of iron, cod-liver oil, fresh air, milk-and-egg diet, and baths are indicated. *Local applications* are useful only after thorough removal of the scales and crusts. This is best accomplished by soaking the lids in a warm alkaline solution. A weak solution of peroxide is also effective. The lids being cleaned, the application of a salve containing 1 per cent. to 4 per cent. of the yellow oxide of mercury, or 1 per cent. of the white precipitate, effects a cure in simple cases. In the *ulcerative form*, besides the removal of crusts, the infected area should be opened and the cilia involved removed by the use of ciliary forceps.

Phthiriasis may be mistaken for blepharitis; but the eggs and the parasites may be detected by close examination. Mercurial ointment effects a cure.

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DISEASES OF THE GLANDS.

Hordeolum.—An *external stye* is the result of a suppurative inflammation of one of Zeiss's glands. Swelling and redness occur over the site of the gland and increase for a few days. After spontaneous or operative evacuation of the contents all inflammatory symptoms rapidly subside.

An *internal stye* is the result of suppurative inflammation of a Meibomian gland. Inasmuch as these glands are larger than those of Zeiss and are imbedded in the dense fibers of the tarsus, an internal stye is more painful and lasts longer than an external one. On everting the lid the pus may be seen through the conjunctiva.

Etiology.—Styes occur in young people, particularly the scrofulous and anemic. Blepharitis causes a stye by the presence of scales and crusts, which serve as a bed for germs which easily enter the glands. The swelling of the margin of the lids may prevent the exit of the secretion of the glands. Errors of refraction, and strain of accommodation may be exciting causes.

Treatment.—Hot applications accelerate the ripening of the abscess and opening by free incision shortens the course of the trouble. As a prophylactic measure treatment of an existing blepharitis is imperative.

Chalazion.—Chalazion is a disease of the Meibomian glands characterized by its chronicity, and the formation of a tumor which causes comparatively little inconvenience, and finally degenerates into granulation tissue and pus. It is easily differentiated from an internal stye, which runs an acute course, is painful, and suppurates.

Chalazia *occur* more frequently in adults than in children. They may produce some deformity and irritate the eye by reason of the chafing caused in winking.

Treatment.—Small chalazia call for no treatment; large ones should be removed. The incision may be made through the skin, conjunctiva, or margin of the lid. The contents should be thoroughly scooped out with a chalazion scoop and the walls of the sac scraped or dissected out. The application of a strong solution of silver nitrate often produces adhesive inflammation of the cyst wall. Cocainization may be sufficient for this operation but the injection of a weak cocain or novocain solution is preferable.

Cretaceous Deposits.—Cretaceous deposits in the conjunctiva sometimes cause irritation. After cocainization they should be picked out. This condition is known as lithiasis conjunctivæ, and is due to degeneration of the contents of a Meibomian gland.

ANOMALIES OF THE EYELASHES.

Trichiasis.—Trichiasis is that condition, not accompanied by inversion of the margin of the lid, in which the cilia are entirely or in part directed inward instead of outward. The irritation of the eyeball causes photophobia and lacrimation. Corneal ulcers and opacities may also occur in consequence of mechanical injury to the cornea.

Etiology.—The most common cause is trachoma, when the trichiasis is often complete—that is, involving all of the cilia. Partial trichiasis, in which only some of the cilia are directed backward, occurs as a result of scars due to blepharitis, stye, injury, or operation.

Distichiasis.—Distichiasis is a rare and generally congenital condition in which an otherwise normal lid has two rows of cilia, one directed forward, the other backward.

Treatment.—When only several cilia are directed inward epilation may suffice. As this does not destroy the follicle, epilation must be repeated from time to time.

The radical treatment is the destruction of the follicle by *electrolysis*. The positive pole of a galvanic battery (eight to twenty elements) is connected with a flat electrode, the negative pole with the needle. The needle is thrust into the follicle by the side of the lash and the flat electrode is placed on the temple. This treatment is practicable only when a few of the cilia turn in. The painfulness of this method militates against its usefulness. When many or all of the cilia are directed inward, epilation cannot be practised. Here the best treatment is an operation to remedy entropion.

ANOMALIES OF POSITION OF THE LIDS.

Entropion.—Entropion is a turning in of a lid. It may be spasmodic or cicatricial.

Spasmodic Entropion.—Spasmodic entropion is caused by contraction of the orbicularis muscle. Two conditions are necessary to produce this kind of entropion: imperfect support of the free border of the lid and excessive relaxation of the skin.

Etiology.—The relaxation of the skin as occurring in the aged, the absence or smallness of the bulb, an eye deeply set in the orbit, blepharospasm, blepharophimosis, or the use of a bandage.

Treatment.—Treatment depends upon the cause. When produced by the use of a bandage, its discontinuance is all that is necessary. If the bandage be indispensable, the inversion of the lower lid may be prevented by the use of a strip of adhesive plaster, one end of which should be stuck to the skin over the edge of the orbit. The use of an artificial eye prevents entropion when caused by absence of the bulb.

Cicatricial Entropion.—Cicatricial entropion is caused by cicatrical shortening of the conjunctiva whereby the free border of the lid is turned inward.

Etiology.—Trachoma, diphtheritic conjunctivitis, and injuries of the conjunctiva.

The *operation* for the relief of entropion will be considered in the section on Operations.

Ectropion.—Ectropion is an eversion of the lid whereby a part of the conjunctival surface is exposed. Eversion of the *puncta* occurs in consequence of the eversion of the margin of the lid, and hence lacrimation is a prominent symptom. The irritation of the secretions causes redness and thickening of the lid margins and hypertrophy of the conjunctiva. High degrees of ectropion produce lagophthalmos, which may lead to corneal disease.

Etiology.—The etiology of ectropion is various. It may be due to spasm which everts the margin of the lid when the tarsal portion is slightly turned from the eyeball and the integument is tense. Protrusion of the eyeball favors spasmodic ectropion. Paralysis of the orbicularis, because of the relaxed condition of the lower lid, may cause paralytic ectropion. In old age the loss of tone of the orbicularis and chronic catarrh of the conjunctiva favor senile ectropion. When scars occur in the integument the result of injury, operation, burns, and the like, cicatricial ectropion results.

Treatment.—In *spasmodic ectropion* the lids should be replaced and a firm bandage applied. *Paralysis of the facial nerve* requires the use of electricity. When the ectropion exists in high degree, surgical means are necessary to give relief.

Symblepharon.—Symblepharon is the adhesion of the palpebral to the ocular conjunctiva in consequence of injuries causing excoriation or sloughing of the opposing surfaces. The adhesion may be complete, in which case the cul-de-sac is obliterated; or partial, when it remains free, permitting the passage of a probe under the bridge of tissue along the fornix between the lid and bulb.

Etiology.—Burns, corrosion, diphtheritic conjunctivitis, trachoma, operations, and ulcers of all kinds may produce symblepharon.

Treatment.—The object of the treatment is to break up the adhesions and prevent union of the exposed surfaces. This can be accomplished in cases of partial symblepharon, but is very difficult or impossible in complete ones. In some cases the employment of skin grafts is useful but the failures are many.

Ankyloblepharon.—Ankyloblepharon is the adhesion of the margins of the lids, the canthi not being involved.

Etiology.—The etiology is the same as for symblepharon, which generally complicates this condition. Two opposing surfaces being denuded by reason of burns, etc., may become adherent.

Treatment.—The treatment consists in permanent separation of the lid margins after a dividing cut has been made. The success of the treatment will depend upon the extent of the symblepharon when it coexists.

Lagophthalmos.—Lagophthalmos is a condition in which the lids cannot be completely closed. It may be due to shortness of the lids, ectropion, paralysis of the orbicularis, absence of corneal reflex, and enlargement or protrusion of the bulb.

Treatment.—The treatment depends upon the cause. When the lagophthalmos is due to shortness of the lids, a canthoplasty is necessary. Lagophthalmos is relieved by an operation for ectropion when this causes it. Paralysis of the orbicularis, which is supplied by the facial nerve, may be due to a variety of causes, such as injury to the nerve, exposure to cold, or intracranial diseases. It can therefore not always be treated. When the lagophthalmos is only a temporary condition, the eye should be bandaged to protect the cornea from injury and irritation. When, however, the condition is not likely to yield to treatment, a *tarsorrhaphy* should be performed to prevent certain destruction of the eyeball.

Blepharophimosis.—Blepharophimosis is the condition in which the palpebral fissure is shortened, either as a result of contraction of the lids or adhesion of the opposing margins of the lids at the outer canthus. It may be congenital, associated or not with ptosis but frequently complicates trachoma. A *canthoplasty* is the treatment.

Blepharochalasis.—Blepharochalasis is a condition common in the aged, due to an excess of lax skin, in consequence of which a fold hangs down in case of the upper lid below the margin. The treatment when desired is excision.

DISEASES OF THE MUSCLES OF THE LIDS.

Spasm and *paralysis* of the orbicularis frequently occur.

Spasm, or *blepharospasm*, as it is called, may be a symptom of many ocular diseases in which there is irritation. Refractive errors, foreign bodies in the conjunctival sac, trichiasis, or whatever disease may be the cause, should receive attention. Blepharospasm occurs also as a disease of itself. It is observed in hysterical young women, and also in old age, when the lids may be quite closed or continually winking. When attempts are made to raise the upper lid there is considerable resistance, which differentiates this trouble from ptosis. When of hysterical origin, blepharospasm often passes away without treatment. In the aged treatment is of no use.

Paralysis of the Orbicularis.—Paralysis of the orbicularis is due to a lesion of the facial nerve; this is more often peripheral, seldom central. Exposure to cold, rheumatism, traumatism, disease of the middle ear or temporal bone, neoplasms, and syphilis are the most common *causes*.

Treatment.—The electric current is of use in all forms, in conjunction with the special treatment that may be indicated. The eye should always be bandaged, to protect the cornea, in severe cases; at night only in simple cases. When there is no prospect of recovery a tarsorrhaphy should be made to close the lids partially.

Ptosis.—Ptosis is a paralysis of the levator palpebræ superioris. It may be congenital, when it is usually bilateral; or acquired, when it is usually unilateral. The upper lid droops more or less, interfering with the sight when the margin of the lid is below the level of the pupil. Lesions involving the muscle or the third nerve are the causes of acquired ptosis. It is often associated with paralysis of the ocular muscles supplied by the third nerve. Ptosis is sometimes seen in the paralytic form of alcoholism.

Treatment.—The treatment depends upon the cause. In some cases surgical procedure is the only means of help.

Sympathic ptosis is due to involvement of the sympathetic which supplies the superior papebral or Mueller's muscle. Although rarely spontaneous, it was occasionally seen after excision of the superior cervical sympathetic ganglion. It is associated with a contracted pupil.

THE LACRIMAL APPARATUS.

ANATOMY.

The lacrimal gland, the accessory lacrimal glands, and the tubular acinous glands of the conjunctiva constitute the *secreting part* of the lacrimal apparatus; the lacrimal canals, the lacrimal sac, and the nasal duct, the *conducting part.*

The **lacrimal gland,** which secretes the tears, is lodged in the lacrimal fossa under cover of the external angular process. It is divided into two parts, a larger (or upper), and a lower, the *accessory gland.* The secretions of the upper gland pass through the ducts of the accessory gland and finally empty into from seven to twelve narrow ducts which pierce the outer side of the superior fornix of the conjunctiva.

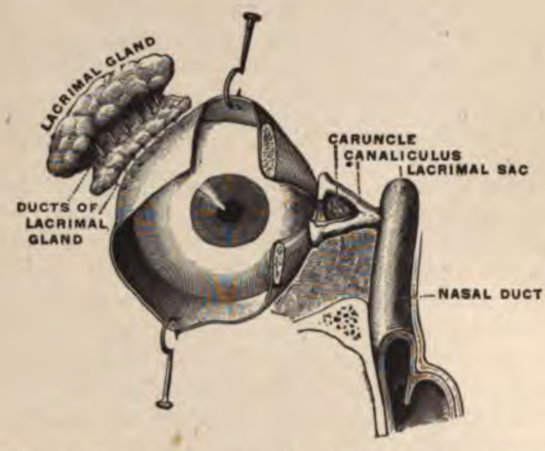

Fig. 16.—The lacrimal apparatus of the right eye. (Testut.)

Only a small amount of the tears is secreted by the lacrimal and accessory glands, the greater part coming from the *tubular acinous glands* of the conjunctiva. The lacrimal gland can accordingly be extirpated without evil consequences, the conjunctival glands supplying secretions sufficient to moisten the surface of the eyeball. The lacrimal gland is supplied by the lacrimal artery, a branch of the ophthalmic. The lacrimal nerve, a branch of the fifth, and branches of the sympathetic supply the gland.

The **lacrimal canals** collect the tears when excreted to excess. These begin at the puncta lacrimalia, which are opposite each other on the free border of the eyelids near the inner canthus, turning toward the eyeball, and lead to the **lacrimal sac.** This lies in a groove at the lower inner angle of the orbit, and gradually merges into the **nasal duct,** which opens into the inferior meatus about one-half inch behind the anterior end of the inferior turbinal.

The lacrimal, superior maxillary, and inferior turbinated bones form the *bony canal* of the nasal duct, which is lined, from within outward, by mucous membrane, a layer of veins, and a fibrous sheath. Temporary engorgement of the veins may interfere with the passage of a probe. When the lacrimal canals and nasal duct cannot accommodate the excess of tears these flow over the lower lid and down the cheek.

CONGENITAL MALFORMATIONS OF THE LACRIMAL APPARATUS.

The lacrimal glands may be **absent,** when there is usually other malformation, as anophthalmos or cryptophthalmos. When children do not shed tears, the lacrimal gland may be present, but the *tubular glands* of the conjunctiva are probably absent.

Dislocation of the lacrimal gland is sometimes congenital, and **fistulæ** have also been observed.

The lacrimal canals sometimes **fail to unite,** when a groove instead of a canal results.

At times the **canaliculi** are but **partially closed;** or they may be wanting.

Atresia of the puncta may be congenital.

A **fistula** of the lacrimal sac or of the nasal duct is a rare occurrence, but **stricture** of the latter is more frequently observed.

NEOPLASMS OF THE LACRIMAL APPARATUS.

Tumors of the lacrimal gland are rare. Adenomas, myxomas, myxosarcomas, sarcomas, epitheliomas, cysts, dermoid tumors, and angiomas have had their origin in the gland.

The removal of the gland should be performed early when it is possible to save the eye. When the growth has involved too much tissue, enucleation is imperative.

Polyps have been met with in the lacrimal canal. They should be removed.

INJURIES OF THE LACRIMAL APPARATUS.

Injuries about the orbit sometimes produce a *fistula of the lacrimal gland*, allowing the tears to escape through the opening in the integument. The fistula should be brought into communication with the conjunctival sac, after which the integument may be freshened and the external opening closed.

Dislocation of the lacrimal gland may be due to traumatism or may occur spontaneously.

Wounds involving the puncta, the lacrimal canals, and the nasal duct may produce *atresia* of any or all of these parts. The possibility of restoring the function depends upon the nature of the injury.

For *atresia of the punctum*, a dilator should be frequently used after the punctum has been opened. If the *canaliculi* are closed, it is necessary to open them with a canaliculus knife and pass probes.

In cases of traumatic stricture of the *nasal duct* it may be necessary to make an opening with a knife or drill, under general anesthesia, after which the probe should be frequently used. If this fails, a gold cannula is of great service.

DISEASES OF THE LACRIMAL APPARATUS.

On the whole, disease of the lacrimal gland is very rare; disease of the drainage or conducting apparatus, on the other hand, is quite common.

Dacryo-adenitis.—Dacryo-adenitis, or inflammation of the lacrimal gland, is a very rare disease. For an acute inflammation the application of heat and an incision as soon as fluctuation is demonstrable are indicated. Antisyphilitic or antirheumatic remedies are useful in the treatment of the chronic form in conjunction with local stimulating applications.

Dacryops.—Dacryops or cyst of the lacrimal gland, is the result of closure of one or more of the excretory ducts, whereby the retention and accumulation of secretion produce a transparent elastic swelling.

Treatment.—The treatment consists in establishing a communication between the cyst and conjunctival sac.

Chalky Concentrations.—Chalky concentrations sometimes form in the lacrimal gland. As soon as they cause irritation they should be removed.

Lacrimal Blennorrhea. — **Definition.** — This is a chronic disease of the mucous membrane of the canaliculi, the lacrimal sac, and nasal duct.

Etiology.—Uncorrected errors of refraction may cause simple cases. Exposure to cold, causing a conjunctivitis or rhinitis, is also an etiological factor in some cases. Hypertrophic or atrophic rhinitis, syphilitic or tuberculous ulcers and tumors, and polyps in particular, are nasal conditions which may lead to stenosis and inflammation of the drainage-apparatus.

Symptoms.—Because of the swelling of this mucous membrane the drainage of the tears is interfered with, and accordingly *epiphora*, or overflow of tears, results. In light cases this may be the only symptom.

As the disease progresses a *stricture* occurs in the drainage apparatus, in consequence of which the lacrimal sac increases in size from retention of secretion. This becomes infected, and soon the lacrimal sac on pressure empties through the canaliculi a mucopurulent secretion.

Complications.—Lacrimal blennorrhea often causes chronic conjunctivitis from irritation of the mucous membrane by the purulent secretion, and ulcerating blepharitis from overflow of tears. Finally this disease leads to eczema, shortening of the lid, and entropion.

Treatment.—The treatment depends upon the cause. When due to syphilis, the appropriate treatment not only relieves lacrimal disease, but prevents caries and necrosis of the lacrimal bone. Nasal diseases should receive attention when they are causative. In all cases the patient should be instructed to press out frequently the purulent secretion;

and cleanliness is to be enjoined. In some cases the passage of the probe accomplishes a cure in a few months; but very often the physician is not justified in promising much.

For passage of the probe, the punctum must be large enough to permit its introduction into the canaliculus, which must also in turn be large anough to allow the passage of the probe into the sac. The punctum can be dilated with a pointed probe; the canaliculus must sometimes be cut before using the probe, but this should not be done until patient and repeated trials fail. For this purpose a canaliculus knife is pushed through the canaliculus until the resistance of the lacrimal bone is felt, when the cutting edge of the knife should be brought upward and inward.

The lacrimal syringe is of use in most cases, but should never be used immediately after any cutting operation, for fear of producing infiltration of the tissues. A saturated solution of boric acid or a 1 to 5000 solution of bichloride is generally used in the lacrimal syringe. After such a solution has been used for a time it is well to substitute 1 to 5 grains of zinc sulphate to the ounce. Never use argyrol or other staining solution, as infiltration may occur.

Dacryocystitis.—Dacryocystitis occurs during the course of a lacrimal blennorrhea. The lacrimal sac becomes the seat of a violent inflammation, which manifests itself by pain, redness, and swelling of the lids. The constitutional reaction is at times great, so that the disease may be confounded with facial erysipelas by the inexperienced. If left alone, a spontaneous opening, known as a *lacrimal fistula,* results.

If seen at the beginning, the passage of a probe and antiseptic injections may abort the disease; but when a lacrimal abscess is imminent it is better to encourage its formation by hot applications. When ripe an incision should be made to evacuate the pus. So long as the lacrimal fistula remains, the danger of a new attack of dacryocystitis is *nil.* After the inflammatory signs have subsided, the drainage-apparatus should receive attention, and the fistula should not be allowed to close so long as there is a stricture in the nasal duct. If the nasal duct cannot be kept open, the lacrimal sac should be excised. This prevents a recurrence of the dacryocystitis;

and as removal of the lacrimal sac removes a source of irritation, the lacrimation is much diminished or even abolished.

The destruction of the lacrimal sac should be undertaken in syphilitic or strumous patients when there is caries of the lacrimal bone.

THE CONJUNCTIVA.

ANATOMY.

The **mucous membrane** forming the inner surface of the lids and the covering of the anterior half of the bulb from the equator to the corneal margin, is the **conjunctiva** (Fig. 17).

The **limbus** is that part of the conjunctiva which marks the corneal margin where the epithelial layer of the conjunctiva becomes transparent and begins to form the anterior layer of the cornea.

The conjunctiva consists of palpebral and ocular parts, which are connected by intervening folds, each known as the fornix.

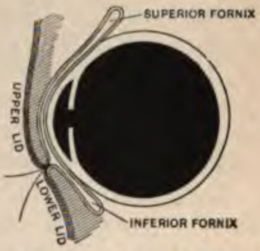

Fig. 17.—Sagittal section of eye, showing superior and inferior fornices of the conjunctiva. (Testut.)

The **palpebral conjunctiva** begins at the free margin of the lid, forming a continuation of its integument. It is also continuous with the lining membranes of the Meibomian glands. Dense cellular tissue firmly unites the tarsal cartilage and underlying conjunctiva, which, because of its thinness, makes it easy to see the outline of the Meibomian glands. Disease of the nasal mucous membrane sometimes affects the conjunctiva, since the lining membrane of the lacrimal canaliculi and sac leading to the nasal duct is a continuation of the conjunctiva.

The **ocular conjunctiva** is that part loosely connected to the bulb by connective tissue. It is very thin, is freely movable over the bulb, and contains no glands and few papillæ.

The crescentic fold of conjunctiva at the inner canthus is the **plica semilunaris**, in which rests partly the **caruncula lacrimalis**. This contains various glands which secrete a whitish substance frequently seen at the inner canthus.

A **cul-de-sac** is formed in the upper and lower lids by the reflection of the corresponding **fornix**. The *superior cul-de-sac* is the deeper, but is difficult to examine without a double eversion of the upper lid. The *lower* is easily seen on requesting the patient to look upward, the lower lid being at the same time held down.

The **fornix** is the thickest part of the conjunctiva, and because of its rich blood supply and laxity of structure takes on great swelling in inflammations of the conjunctiva. The fornix contains lymphatic follicles and accessory lacrimal glands (Krause's).

The *superior* cul-de-sac contains numerous tubular acinous glands, the secretion of which is similar to that of the lacrimal gland. It is from this source that the eye is constantly kept moist, the lacrimal gland functioning only at times, such as in crying. The presence of the lacrimal gland is accordingly not very important.

The fornix, because of its elasticity and folds, permits the needful motility of the bulb.

Function.—It forms the smooth inner surface of the lid, and its glands furnish secretions which lubricate the parts, thus facilitating movements of the lids and eyeball without friction, and which moisten the cornea, thus preserving its clearness and integrity.

CONGENITAL ABNORMALITIES OF CONJUNCTIVA.

Subconjunctival fatty growths, nevi, tumors composed of bone tissue, and dermoid tumors may be congenitally present in the conjunctiva.

Dermoid Tumors.—Dermoid tumors most frequently occur, and are often associated with colobomas of the iris and the choroid. The sclerocorneal junction is the usual seat of these growths, and the cornea is more or less involved.

Treatment.—When, for cosmetic reasons or inconvenience, the patient seeks help, the treatment is removal.

NEOPLASMS OF THE CONJUNCTIVA.

Granulomas occur in the wake of healing wounds and injuries of the conjunctiva. Foreign bodies imbedded in the cul-de-sac frequently cause them.

A **polypus** resembles a granuloma, which can be distinguished from it by the absence of the epithelial covering which the polypus always possesses. Polypi usually occur on the caruncle.

Papillomas often spring from the caruncle, but may also be found in the palpebral conjunctiva.

Treatment.—*Granulomas*, *polypi*, and *papillomas* should be removed and the stumps cauterized when broad.

Besides these, cysts, angiomas, fibromas, myxomas, and osteomas occur in the conjunctiva.

Epitheliomas and **sarcomas** are the malignant growths which involve the conjunctiva. They occur in old age, and unless radically removed in the earlier stages the patient succumbs. These growths usually spring from the limbus of the conjunctiva and spread over both cornea and conjunctiva.

Treatment.—When these malignant growths cannot be thoroughly removed by use of the knife, curette, and cautery, and the *x*-rays, radium or electrical desiccation are without avail, the eye should be *enucleated*, even though vision may not be affected.

INJURIES OF THE CONJUNCTIVA.

Foreign Bodies.—Dust, cinders, ashes, and small particles of similar substances often lodge on the eyeball, and, on winking, are carried away, only to become again lodged in the palpebral conjunctiva of the upper lid, usually in the subtarsal fold. The *pain*, which is sometimes very severe, is due to the chafing of the cornea by the foreign body during winking.

Treatment.—On everting the lid the foreign body is easily removed. Great mischief is sometimes done by the patient, who, at the suggestion of friends, applies raw steak or poultices of various kinds, producing serious infection.

Foreign bodies of *considerable size* occasionally lodge in the fornix, and may remain there for some time without inconvenience.

Wounds.—Wounds of the conjunctiva are usually attended with ecchymosis. They should be *treated* on surgical principles.

Corrosion and Burns.—Corrosion and burns of the conjunctiva often occur as the result of contact of acids, caustic alkalies (lime), steam, hot water, molten metal, and the like.

Symptoms.—Not only is there loss of conjunctival tissue, but also frequently *symblepharon*, which more or less interferes with the motility of the eye. The impairment of sight depends upon the severity of the injury to the cornea.

Treatment.—The treatment consists in removal of the corrosive substance as soon as possible, and attempts to neutralize or render it inert. When injury is due to *caustic alkalies*, milk instead of water should be used to flush the conjunctival sac. Injury due to *lime* is best treated first with application of oil and then with instillations of a saturated solution of cane-sugar.

The *inflammations* produced by burns and corrosion are best treated with cold applications, atropine, and the bandage.

In *severe injuries* the lids should frequently be lifted from the bulb, in order to prevent adhesion of the denuded surfaces and sterile vaseline freely used in the conjunctival sac.

When *symblepharon* does occur, an operation must be done to relieve to some extent the adhesions which prevent motility of the bulb.

DISEASES OF THE CONJUNCTIVA.

Acute Catarrhal Conjunctivitis.—Definition.—This is an inflammation of the conjunctiva lasting in uncomplicated cases from one to two weeks.

Its *extent* varies with the severity of the disease, simple cases involving the palpebral conjunctiva and fornix; severer ones, the conjunctiva of the bulb as well. There is hyperemia of the mucous membrane and the secretion of mucus is increased.

Symptoms.—The patient complains of photophobia and the sensation of a foreign body in the conjunctival sac, which is produced by the presence of stringy mucus. Burning and itching of the lids occur, but there is no great pain unless the cornea and iris be involved. The secretions dry upon the edges of the lid during the night, gluing the lids together in the morning.

Etiology.—The usual cause is bacterial and the Koch-Weeks bacillus and the pneumococcus are important. Less frequently observed are the diplobacilli which often produce chronic conjunctivitis, the influenza bacilli, the pneumobacilli, the Bacteria coli and the streptococci. The Koch-Weeks bacillus is highly contagious and frequently becomes epidemic.

Treatment.—*Simple cases* of acute catarrhal conjunctivitis require only attention to cleanliness. A saturated solution of boric acid flushed into the conjunctival sac every two or three hours is all that is necessary. Simple cases are sometimes transformed into complicated ones by injudicious treatment. Mydriatics and cocaine should not be instilled; and bandages, poultices, and compresses never be used. Eye-strain predisposes to conjunctival catarrh, and errors of refraction should receive attention in all cases apparently not infective.

Severer cases, as those due to the Koch-Weeks bacillus, are best treated by applications of a 2 per cent. silver nitrate solution, care being taken that the solution does not come in contact with the cornea. This treatment should be carried out daily, besides attention to cleanliness. A solution of argyrol (5 to 20 per cent.), protargol (2 to 10 per cent.), or silvol (5 to 20 per cent.) may be employed by the patient and a drop instilled three times daily after the conjunctival sac has been thoroughly cleaned by the use of any mild wash. The pneumococcus infection is best treated by the instillation of a few drops of a 1 per cent. solution of ethyl hydrocuprein, hydrochloride of quinine. Several drops of this are to be instilled three times daily.

Chronic Catarrhal Conjunctivitis.—This is an affection of adults, particularly in old age.

Symptoms.—The patient complains of various sensations, such as dryness, burning, lacrimation, and fatigue of the eyes, particularly in evenings; and it will be noticed that the conjunctiva is slightly congested, the lid margins thickened in consequence of blepharitis, and a small amount of tenacious secretion accumulates at the inner canthus. The secretion often glues the lids together during the night.

Treatment.—Treatment of this very frequent disease demands attention to errors of refraction, the general health, and regulation, if possible, of the employment, that irritating influences may be removed. Existing troubles of the drainage apparatus should receive the proper treatment; and ectropion, if present, should be remedied. Aside from the special treatment which the particular case demands, the use of mild astringent and antiseptic solutions is indicated. When the conjunctivitis is due to the diplobacillus of Morax-Axenfeld, a solution of the sulphate of zinc (0.5 per cent.) will be found an excellent remedy.

Follicular Conjunctivitis.—**Etiology.**—The cause of the disease is uncertain; but the fact remains that follicular conjunctivitis occurs in anemic or scrofulous individuals who often have refractive errors.

Diagnosis.—The diagnosis between this and *trachoma* is particularly important, because the granules look like those of trachoma.

Histologically, both are seen to consist of adenoid tissue; but the clinical picture of the diseases is very different:

Follicular conjunctivitis occurs in childhood and youth; trachoma is generally a disease of adult life.

Follicular conjunctivitis is not a serious trouble, and often terminates without consequences; trachoma is a serious disease, which often leaves sequelæ interfering with the sight and comfort of the patient. The follicles occur on the surface of the palpebral conjunctiva, and particularly in the fornix of the lower lid, and are easily brought into view on eversion of the lids.

Treatment.—The granules should be expressed after cocainization with the aid of a ciliary forceps. An antiseptic lotion should then be used for a few days and refractive errors corrected. Constitutional treatment is generally needed and should be prescribed.

Gonorrheal Conjunctivitis.—Definition.—This is an infectious disease due to the presence in the conjunctival sac of the *gonococcus.* When the disease occurs in adults, it receives the name blennorrhea adultorum; when infection occurs during the parturient act or during the puerperal state, the newborn infant is said to have blennorrhea neonatorum.

Symptoms.—The symptoms are first those of an acute catarrh; but in a day or two violent inflammation manifests itself by causing great swelling of the lids and conjunctiva. The ocular conjunctiva is swollen to the corneal edge (chemosis), so that the cornea seems sunken or depressed. Whenever during the course of a conjunctival disease, the ocular part shares in the inflammation, a microscopic examination of the secretions should be made to determine the cause. As soon as the discharge becomes free, the pain and heat subside.

Complications.—The complication to be feared is *perforation of the cornea,* which may be due to the macerating effect of the discharge or to interference with corneal nutrition. Corneal ulcerations may heal without having produced perforation, but the vision is greatly diminished when a central opacity remains.

Incarceration of the iris in the corneal opening and panophthalmitis do occur after some perforations.

Treatment.—The *preventive treatment* is very satisfactory. A drop or two of a 2 per cent. silver nitrate solution in the conjunctival sac of each eye after birth of the child reduces the number of blennorrhea neonatorum cases. In all cases precautions should be taken to prevent infection of the other eye.

The *treatment* of both forms is the same, although the disease in adults appears more severe. If seen early, when there are hyperemia, pain, and heat in the affected parts, cold applications by iced cloths give great relief. An abortive treatment is rarely of use, and may be detrimental. A 2 per cent. solution of silver nitrate brushed over the conjunctiva two or three times daily is of value when the inflammation is not severe and discharge slight. When the swelling is considerable and the discharge excessive, stronger solutions may be employed. The cleanliness of the conjunctival sac is of the greatest importance, and to accomplish this a bichloride solution (1 to 10,000); or a saturated boric acid solution may be employed. After the hyperemia has subsided the use of heat instead of cold will be found of service. The periodical application of heat increases the activity of the circulatory and absorbent systems, and would hence be of great value to keep up the nutrition of the cornea, the preservation of which is the end of treatment.

Prognosis.—The prognosis depends upon the physical condition of the patient and implication of the cornea.

To *relieve the pressure* on the cornea a canthotomy may be made; this also facilitates treatment.

When the *cornea is perforated,* the use of iodoform or the tincture of iodine in the opening is recommended.

The *prolapsed iris* should not be snipped off. Cleanliness should be insisted on as before the perforation and atropine be employed.

The *after-treatment* of all cases consists in the use of mild astringent instillations for some weeks.

The conjunctiva after this disease is subject to other affections.

Trachoma.—**Definition.**—This is a chronic disease of the conjunctiva, probably produced by a germ. The disease is a very serious one, for it more or less prejudices the efforts of the patient to make a living, either by interfering with vision or by necessitating prolonged treatment.

Complications.—During the course of the disease, pannus, corneal ulcer, and iritis are frequent complications; and the possible *sequelæ* are distortion of the lids, symblepharon, xerosis of the conjunctiva, and corneal opacities.

The disease appears in two forms: The *papillary,* in which elevations are formed on the surface of the tarsal conjunctiva, more particularly of the upper lid; and the *granular,* in which granules resembling grains of boiled sago develop in the deeper layer of the conjunctiva, affecting the fornix more than the tarsal part. Both forms may occur at the same time,

Symptoms.—The conjunctiva secretes a mucopurulent secretion, which causes the lids to stick together; and the irritation is such that there are usually lacrimation and photophobia. The pain may be considerable when pannus, corneal ulceration, and iritis complicate the disease. The upper lid is not fully opened because of the photophobia and thickness of the lid.

The constant irritation of the cornea by the uneven surface of the conjunctiva frequently causes the growth of a network of bloodvessels, which begins at the periphery and extends to the centre of the cornea: this is *pannus*. It produces an irregularity in the surface of the cornea; and, being superficial, can easily be differentiated from deeper corneal disease. The vessels, moreover, have a bright red color and branch like the limbs of a tree.

Ulceration of the cornea occasionally complicates trachoma, and may occur with or without pannus.

Both corneal ulceration and pannus may cause *secondary iritis.*

Treatment of Trachoma.—Constitutional treatment and attention to cleanliness are of the greatest importance. The patient should be instructed to flush frequently the conjunctival sac with some weak antiseptic solution. The objects of the treatment are to reduce the conjunctival inflammation and the hypertrophy. Cleanliness and weak astringent applications, such as a 1 or 2 per cent. solution of silver nitrate, are of use when the secretion is abundant and inflammation severe. Caustics, friction, and surgical measures may be employed after subsidence of the inflammation. Cupric sulphate is usually employed three or four times weekly as a caustic, care being taken that the cornea is not touched. After eversion of the lids, iodoform gauze containing an excess of powdered iodoform or boric acid may be employed to rub the conjunctiva.

Of the *surgical methods,* expression of the granule after incision of it is frequently employed. Expression may be accomplished with the roller forceps or by pressing the conjunctival surface of the lids together between the thumb-nails. If the patient has a unilateral disease, he should be warned of the danger of infection to the other eye. Not only to prevent infection of the other eye, but to prevent the infection of other individuals, should the patient be careful.

Treatment of Pannus.—This affection frequently disappears during the treatment which is directed to the lids. In some obstinate cases, however, other measures are necessary. An infusion of powdered jequirity bean (15 to 30 grains to the ounce) should be applied in such cases to the conjunctiva three times daily for several days, exciting considerable inflammation, after subsidence of which the pannus will have disappeared. Instead of an infusion, the powdered bean in the quantity of a grain may be applied to the conjunctiva.

Treatment of Ulceration of the Cornea.—Atropine and hot-water fomentations are indicated. Copper sulphate applications to the conjunctiva and the bandage, which would retain the secretions, are *contra-indicated*. *Secondary iritis* will be relieved by the application of heat and instillation of atropine.

The *sequelæ of trachoma* already mentioned demand special treatment. An operation can remove the irritation of trichiasis and restore the normal position of the lid. For xerosis, emollient instillations relieve the dryness due to absence of secretion. Symblepharon is not amenable to treatment. Corneal opacities may be partially removed by use of the salve of the yellow oxide of mercury or a 5 per cent. solution of dionin.

Membranous Conjunctivitis.—**Definition.**—This is a rare disease characterized by the formation of a membrane upon the palpebral conjunctiva of individuals whose general condition is poor.

Clinically this disease appears in two forms, the *croupous* and the *diphtheritic*.

Croupous Conjunctivitis.—*Symptoms.*—This is more common than the diphtheritic, and may be mistaken for it.

The white or grayish membrane can usually be removed without much force; systemic disturbance is slight; pain and swelling of the lids are not great; and the lids are fairly pliable. After spontaneous exfoliation of the membrane takes place, a purulent infectious discharge develops which persists for a short time.

Treatment.—The treatment should consist in cold applications in the hyperemic stages and the use of heat as soon as detachment of the membrane begins. A 0.5 to 1 per cent. solution of silver nitrate should be applied to the part of conjunctiva from which the membrane has been cast off. Cleanliness is to be observed in all stages of the disease.

Diphtheritic Conjunctivitis.—*Symptoms.*—This may be associated with faucial or nasal diphtheria, but is frequently observed alone. There is generally considerable constitutional disturbance, and the picture of the disease is that of an aggravated form of croupous conjunctivitis. The redness and swelling of the lids are extreme, and the pain is great on manipulation of the lids, which become as hard as a board, rendering eversion an impossibility. The ocular conjunctiva may become hyperemic and corneal complications are likely to occur.

Treatment.—In the treatment sustaining measures should be prescribed. When the presence of the Klebs–Loeffler bacillus can be demonstrated, antitoxin suggests itself. Attention to the cleanliness of the conjunctival sac, so far as it does not necessitate rude handling of the board-like lids, should be enjoined. Bichloride solution of the strength tolerated by the patient should be used for its antiseptic effect. The use of heat should be faithfully continued in order to increase the nutrition of the cornea and prevent complications which indicate the use of atropine. When opposing surfaces of the mucous membrane are denuded, the lids should be frequently moved over the surface of the ball to prevent symblepharon. Vaseline may be employed for the same purpose.

Phlyctenular Conjunctivitis.—**Definition.**—This is a manifestation of scrofula, tuberculosis, anemia, or faulty assimilation, especially frequent in *children.*

The lesion is a *vesicle,* which is seen upon the ocular conjunctiva or under the epithelial layer of the cornea. In some cases it seems to be associated with nervous derangement.

The disease varies in severity of the **symptoms**, owing to different susceptibilities of the nervous system and the existence or not of pressure by the exudate.

In some cases the only manifestation is a slight redness of the conjunctiva about the vesicle, producing no other symptoms.

On the other hand, lacrimation, photophobia, blepharospasm, ectropion, eczema of the cheek, excoriation and ulceration of the lower lid, and corneal ulcers may be present.

Many patients, if left alone, bury their heads in pillows or seek dark corners to avoid the light. The appetite is usually poor or capricious.

Treatment.—The treatment depends upon the severity of the symptoms. Constitutional treatment is of prime importance, hematics being indicated. The syrup of iodide of iron is particularly useful. In some cases the salicylates are efficacious. When the symptoms are severe, a thorough examination, even if it require the use of an anesthetic, should be made in order to determine the presence of corneal ulcers, which, if neglected, might end disastrouly. *Blepharospasm* yields to immersion of the little patient's face in cold water. The use of lid retractors or a spring speculum for several minutes a couple of times daily is also of value. *Excoriations* yield to cleansing of the conjunctival sac and the application of zinc salve. *Fissures* of the outer canthus heal promptly after use of the silver nitrate in strong solution or in stick form. *Ulceration of the cornea* is best treated by cleansing the infected surface and cauterization with the galvanic cautery or carbolic acid. The preliminary instillation of cocaine is sufficient for the cauterization when the patient is not intractable.

The antiseptic effect of calomel dusted in small quantity in the eyes; or of a salve of the yellow oxide of mercury (1 grain to the dram) rubbed about the conjunctival sac, is of value. Atropine should be employed when there are corneal complications.

When the disease is very obstinate, change of climate and correction of existing refractive errors are of value. In some cases attention to the nasal mucous membranes effects a speedy cure. When the corneal ulcers are superficial, eserine might be tried when this complication does not yield after use of atropine.

Vernal, or Spring Catarrh.—**Symptoms.**—This is a rather uncommon disease, appearing with the first warm days of spring and lasting until fall, characterized by circumcorneal hypertrophy of the bulbar conjunctiva, and granular elevations of the tarsal portion. The conjunctiva appears uneven and thickened and has a milky appearance.

Treatment.—The treatment, which unfortunately is only palliative, consists in mild astringent instillations and antiseptic lotions.

Specific Ulceration of the Conjunctiva.—This may be of *tubercular* or *syphilitic* origin.

Tuberculous Ulcer.—The tuberculous ulcer may be a primary affection. It has ragged edges, and may from a small beginning involve the entire conjunctiva and cornea. In consequence of extensive ulceration symblepharon may result. Pain is not present unless the cornea be involved. *Treatment* should be both general and local. Cleansing antiseptic solutions should be frequently employed. Small ulcers may be cauterized, but larger ones demand milder treatment. The use of tuberculin (from $\frac{1}{100}$ to $\frac{1}{10}$ of a milligram) in some cases produces almost magical results. This may be injected once weekly for several months if necessary.

Syphilitic Ulcers.—Syphilitic ulcers may represent the primary lesion, in which case the induration and glandular involvement make the diagnosis easy. When the ulcer is soft, recognition is not so easy. Caustics should not be used, but the *antisyphilitic treatment* instituted.

Xerosis of the Conjunctiva.—**Symptoms.**—This disease is characterized by dryness and hardness of the mucous membrane whereby the natural secretions are materially diminished or abolished. It may be the result of cicatricial changes incident to trachoma, traumatism, or diphtheritic conjunctivitis; and of exposure of the conjunctiva, such as occurs in ectropion and lagophthalmos. It may also be *primary*, particularly in children whose health is of the worst. In these cases, besides the dryness and accumulation of mucus, night-blindness and corneal complications occur.

Treatment.—When the general health is at fault, tonics and constitutional treatment may improve the disease. Infants usually die in spite of all treatment. When due to cicatricial changes, the use of emollients is palliative. Cure there is none. When caused by ectropion or lagophthalmos, operative interference is of value.

Pterygium. — Definition. — Pterygium is a triangular-shaped, vascular thickening of the bulbar conjunctiva, which finally invades the cornea, occurring usually over the situation of the internal rectus. The apex or head of the pterygium is near or upon the cornea, the base toward either the inner or the outer canthus, according to the side from which it starts. A pterygium may for a long time remain stationary or grow until its apex covers the pupil, interfering seriously with vision. A stationary pterygium is thin, pale (because it is not very vascular), and tendinous in appearance. When *progressive*, it is thick and the edges alone are whitish and non-vascular.

Etiology.—The etiology is obscure, but irritation seems to be a factor.

Treatment.—A progressive pterygium should be removed without delay. A stationary one when there are no symptoms may be left alone. For removal of the pterygium the ligature, transplantation, or excision may be employed. When very large, so that after excision it is impossible to coapt the edges of the conjunctiva, the use of skin-grafts is of value.

Subconjunctival Hemorrhage.—**Etiology.**—When involving the bulbar conjunctiva particularly, it occurs after operations such as tenotomies; and is sometimes a manifestation of conjunctival disease. During the course of whooping-cough it is not uncommon. Sneezing and vomiting as well as coughing may cause it. In old people, in whom the walls of the bloodvessels have degenerated, rupture may occur, producing subconjunctival hemorrhage. Fracture at the base of the cranium sometimes permits the blood to find its way into the orbit, thus showing itself under the conjunctiva.

Treatment.—Treatment is given more to satisfy the patient than to hasten the absorption. Any placebo will answer the purpose. In the aged, if the blood-pressure is high, the cause should be ascertained and general treatment instituted.

Chemosis.—Chemosis, or *edema of the conjunctiva*, the ocular part particularly, is usually symptomatic. In severe inflammations of the lids or orbital tissues, glaucoma, conjunctivitis, iritis, or iridocyclitis, it may be a prominent symptom. Idiopathic chemosis does, however, occur, probably resulting from a disturbance of the circulation. Chemosis at times causes an elevation of the conjunctiva around the cornea, which appears sunken. The color of the thickened conjunctiva is not like that of the dark red extravasation observed in subconjunctival ecchymosis, but is of a pale pink or yellow.

Treatment.—To relieve the edema, punctures or incisions may be called for when the nutrition of the cornea is interfered with. Applications of hot compresses facilitate the absorption of the fluid.

THE SCLERA.

ANATOMY.

The sclera and cornea form the **outer coat** of the bulb, the sclera representing the posterior five-sixths.

The optic nerve enters the posterior part through a perforation known as the **foramen scleræ.**

The **external scleral surface,** which is smooth, with the exception of the insertions of the recti and oblique muscles, is connected to the *capsule of Tenon* and the *ocular conjunctiva.*

The **internal scleral surface** is loosely connected to the *choroid* and *ciliary body* by the *lamina fusca.*

The **outer layers** of the sclera reinforce the outer layers of the optic nerve sheaths.

The **middle layers** of the sclera cross the entrance of the optic nerve, forming the *lamina cribrosa*, the numerous perforations of which transmit the optic nerve fibers.

Through the large central opening, the **porus opticus,** passes the *arteria centralis retinæ.*

The cornea and sclera are similar in structure, the only difference being an **opacity** of the fibrous connective tissue of the *sclera*.

The sclera receives its **blood supply** from the long, short, and anterior ciliary arteries. The anterior ciliary arteries pierce the sclera a short distance behind the corneal margin. These also supply the iris and ciliary body, and form largely the *anterior ciliary zone*.

Inflammation of the iris, ciliary body, and choroid produces engorgement of this zone, and hence the **anterior ciliary zone** is then easily seen just back of the sclerocorneal junction. Congestion of the anterior ciliary zone is easily differentiated from that of the ocular conjunctiva by producing pressure of the lid on the bulb, when the conjunctival congestion temporarily subsides, while the other remains unchanged.

Schlemm's canal, or *circulus venosus*, is a sinus situated at the sclerocorneal junction near the internal surface of the sclera. It is in communication with the anterior chamber, and empties its contents into the anterior ciliary and scleral veins. The drainage of the eye is effected through this canal, and hence *glaucoma* can be caused by conditions which interfere with the function of this canal.

Congenital Pigmentation.—Congenital pigmentation of the sclera is not uncommon and might be mistaken for pigmented patches of the conjunctiva.

Neoplasms.—Neoplasms of the sclera are very rarely primary; but secondary involvement frequently occurs. Fibromas, sarcomas, and osteomas primarily occurring in the sclera have, however, been observed.

INJURIES OF THE SCLERA.

Contusions.—Contusions of the sclera may be of serious import to the eye, since intra-ocular complications frequently coexist. When the contusion is very violent and the elasticity of the eyeball insufficient, rupture of the globe results. The lens and iris may be expelled when the conjunctiva is torn. Rupture usually occurs at the inner and upper margin of the cornea, and the eye is generally lost, although attempts to save the eye may at first seem feasible. Panophthalmitis frequently occurs, and at times it is necessary to enucleate the injured eye when pain and irritation suggest a possibility of sympathetic inflammation.

Burns.—Burns may affect the sclera.

Wounds.—Wounds of the sclera vary in kind and severity, but are very serious when a puncture is made by any unclean agent.

When the *ciliary body* is injured in consequence of a wound of the sclerocorneal margin, sympathetic inflammation and iridocyclitis are likely to occur. The danger of the injury is increased in all cases when the *retina, choroid,* or *vitreous* is involved.

Foreign Bodies.—Foreign bodies seldom lodge in the sclera: they either rebound from the eye or pass through the globe. Foreign bodies in the eye are always a menace, and should, if possible, be removed. Particles of iron or steel may occasionally be removed by the use of an electromagnet, and the attempt should be made, if there is any possibility of saving the eye. Very often it is an act of charity to enucleate an eye containing a foreign body, since the patient is thereby spared great suffering and the possible loss of the uninjured eye from sympathetic processes.

DISEASES OF THE SCLERA.

Episcleritis.—**Etiology.**—The cause of episcleritis probably lies in the tendency to gout or rheumatism or the existence of syphilis or tuberculosis, and when this has been determined, the proper treatment should be instituted.

Symptoms.—This is a chronic disease with a tendency to recurrence affecting the external layer of the sclera in such a way that a sharply limited elevation near the limbus develops. This is not movable, and pressure over the surface does not cause the complete emptying of the bloodvessels seen in conjunctival congestion. The portion of the conjunctiva over the affected area is somewhat congested, but there is little or no discharge. Lacrimation, photophobia, and pain sometimes of a severe type are the prominent symptoms.

Diagnosis.—It may be mistaken for phlyctenular conjunctivitis at the beginning; but the fact that it is a disease of adults more particularly, whereas children are more often subject to phlyctenular troubles, should be kept in mind.

Local Treatment.—Local treatment consists of scarification at its incipiency, and the use of dry heat and atropine. Subconjunctival injection of a normal salt or bichloride solution (1 to 1000) is of use in some cases.

Scleritis.—Scleritis is a chronic disease in which the deeper layer of the sclera is involved, in consequence of which the ciliary body, iris, choroid, and cornea are likely to become secondarily affected.

A thickening of the sclera takes place at the site of the inflammation, and after the disease has run a tedious course, absorption of the exudate takes place, leaving the sclera much thinned, so that when the intra-ocular tension is increased a bulging, or *staphyloma,* as it is termed, results.

Etiology.—The causes of scleritis are uncertain, yet manifestations of syphilis, rheumatism, gout, or tuberculosis are frequently evident. Females are more frequently affected than males, and menstrual difficulties may have some causal influence.

Complications.—Scleritis may cause opacities of the cornea, seclusio pupillæ (complete circular adhesion of the iris to the lens), opacity of the lens or vitreous, myopia, and secondary glaucoma.

Treatment.—The treatment consists in attention to the general health and local application of hot cloths. Rest is absolutely necessary in some cases, and the use of atropine is indicated in ciliary and iritic complications. An iridectomy is indicated when there is no communication between the anterior and posterior chambers, in order to prevent secondary glaucoma. The operation should not be undertaken until inflammatory manifestations have subsided.

Ectasia.—**Partial ectasia** of the sclera is a bulging due to thinning, increase of intra-ocular pressure, or the combination of these conditions, and may occur at the anterior part of the globe, the equator, or the posterior part.

Complete ectasia involves the whole eyeball, and is either *congenital* or occurs in *infancy.*

When *congenital,* the filtration angle is either absent or reduced in size because of adhesion of the iris and the cornea; and the sclera, being elastic in infancy, yields to the increased pressure induced by the interference with the drainage through Schlemm's canal.

An **anterior ectasia** or **staphyloma** involves either the sclera covering the ciliary body (*ciliary staphyloma*) or that immediately in front of it (*staphyloma intercalare*). The *cornea* is always more or less displaced.

Posterior staphyloma may be due to posterior sclerochoroiditis. When congenital it causes a malignant form of myopia. Myopia of high degree—more than 10 D.—is also associated with a posterior staphyloma.

The **treatment** of *anterior* or equatorial *ectasis* is an iridectomy. Pain and cosmetic reasons demand an enucleation when other means of help fail.

Posterior staphylomas are not amenable to treatment, but the coexisting myopia should be corrected.

Complete ectasia, which is known as *hydrophthalmos* or *buphthalmos,* is sometimes progressive, so that the eyeball assumes enormous dimensions, and the sight is lost.

Treatment.—Treatment is of no avail in such a case, and when the condition is stationary, interference is not necessary.

THE CORNEA.

ANATOMY.

The cornea and sclerotic represent the **fibrous envelope** of the eye (Plate II). The cornea forms the anterior transparent one-sixth, the sclerotic, the posterior five-sixths of the eyeball. There is no abrupt line of demarcation between them. The cornea lies in the sclera, which externally slightly overlaps its margin. The cornea is ellipsoidal in shape, the horizontal diameter being slightly longer than vertical.

The cornea is divided into five layers, from without inward, as follows: (1) epithelial layers; (2) anterior limiting membrane, or membrane of Bowman; (3), true corneal substance; (4) the posterior limiting membrane, or elastic lamina; and (5) the endothelial layer.

The **epithelial layer** consists of three kinds of epithelial cells, squamous externally, then polyhedral, and columnar internally.

The **membrane of Bowman** is a fibrillate dense layer which is like the corneal layer proper, except that it contains no corneal corpuscles.

The **true corneal substance** constitutes the larger part of the cornea and is one-twenty-fifth of an inch in thickness. It consists of a *ground substance*, made up of lamellæ which cross each other at different angles, united by an interfibrillar *cement substance;* and of *corneal corpuscles*, which occupy the space between the lamellæ.

The *interlamellar spaces* are connected with one another by narrow canals.

The **posterior limiting membrane**, or Descemet's, is a thin membrane intimately connected to the posterior surface of the true corneal substance. It is the toughest of the corneal layers, and is quite brittle. It is continuous with the pectinate ligament of the iris at its margin.

The **endothelial layer** is a single stratum of polygonal flattened cells which lines the internal surface of the posterior limiting membrane.

The long and short **ciliary nerves** divide into about forty branches, which pierce the sclera to enter the cornea at its periphery.

The cornea has no bloodvessels, but is exceedingly rich in nerves, which do not interfere with its transparency, because they have no sheaths. The nerves form two networks: the one supplying the membrane of Bowman and the external epithelial layer; the other the posterior layers of the cornea. The anterior denser network is situated just behind Bowman's membrane, the posterior delicate network is just in front of Descemet's membrane.

The cornea gets its **blood supply** from episcleral branches of the anterior ciliary arteries and from the lacrimal and infratrochlear arteries which supply the conjunctiva. These divide into a capillary network in the true corneal substance at the periphery of the cornea, and the nutrient material passes through the canalicular lymph system to all parts of the cornea.

The cornea has a greater **refractive power** than any other part of the dioptric system of the eye, and hence claims great attention in correction of refractive errors. The *index of refraction* of the cornea is 1.3523.

CONGENITAL ABNORMALITIES OF THE CORNEA.

Macrocornea.—Macrocornea is a condition in which the cornea is enlarged, occurring in buphthalmos.

Microcornea.—Microcornea is a small cornea, and is usually seen in microphthalmic eyes.

Opacities.—Opacities of the cornea may be congenital and vary in size and intensity. Complete opacities are more often associated with buphthalmos, although they do sometimes occur in microphthalmic eyes. Partial opacities show great variations of size and degree.

Conical Cornea.—Conical cornea, or *keratoconus*, may be congenital, although its development sometimes extends over many years. The cornea protrudes and is conical in shape, the apex being usually the thinnest part and subject to opacity. The increased length of its axes renders the eye myopic, and the irregular curvature of the cornea produces astigmatism.

Examined with the *ophthalmoscope*, one observes a central red reflex which is surrounded by a circular shadow. *Treatment* is of little use.

Dermoid Growths.—Dermoid growths of the cornea are invariably congenital and involve more or less of the ocular conjunctiva. They are usually associated with a coloboma of the lid or other anomaly.

When they irritate the eye, their removal is necessary. This should be thoroughly done and the conjunctiva brought over the denuded conjunctival surface. Where too much conjunctiva has been removed, skin-grafts may be employed as in treating very large pterygia.

Neoplasms.—Neoplasms rarely occur primarily in the cornea. Carcinomas and sarcomas starting in the limbus of the conjunctiva invade the cornea secondarily.

INJURIES OF THE CORNEA.

Foreign Bodies.—Foreign bodies of endless variety may lodge upon or in the cornea. After cocainizing the eye these should be removed. When *superficial*, the spud is the best instrument; when deep, the needle is best suited to the case.

When there is danger of pushing the foreign body into the anterior chamber, the lids should be kept open by a speculum, the eyeball held by a fixation forceps, and a Beer cataract knife pushed through the cornea behind the foreign body. With this as a guard, attempts at removal may be safely made.

A mild antiseptic wash is useful in most cases after removal of the foreign body, and occasionally, when there is considerable reaction, atropine may be necessary to rest the eye.

Injuries of the cornea of whatever kind are serious according to their depth and location. When only the epithelial layer is involved, no scar results, which does occur when the subsantia propria is injured. A central injury producing a central opacity impairs the sight according to its intensity.

Contusion.—Contusion of the cornea results in a loss of epithelium, which after a few days' rest is regenerated.

Wounds.—*Penetrating wounds* of the cornea are serious according to the location and complications. Wounds at the sclerocorneal junction, from possible injury to the ciliary body, leading to sympathetic trouble and prolapse of the iris, are very dangerous.

Traumatic cataract, dislocation of the lens, prolapse of the iris or vitreous, or a coincident injury of the choroid and retina may complicate a corneal wound.

When infected, the danger of corneal wounds is increased, so that the outcome may be a panophthalmitis.

Treatment of Injuries.—In simple wounds cleanliness is all that is necessary. When there is danger of iritis, atropine should be used. A prolapsed iris should be snipped off.

Foreign bodies penetrating the eyeball more frequently pass through the cornea than the sclera. The wound can be seen by oblique illumination. The object may remain in the anterior chamber, become imbedded in the lens with or without wounding the iris, pass through the lens or zonula, and either remain in the vitreous chamber or penetrate the coats of the eye. The danger depends upon the presence of infection and damage done to the structures. A foreign body should in every case be removed.

Burns and Corrosion.—Burns and corrosion of the cornea frequently occur. When superficial, they cause no impairment of sight; when deep, the cornea may become opaque.

Treatment.—The treatment consists in cleanliness of the conjunctival sac, the instillation of atropine, and rest.

DISEASES OF THE CORNEA.

General Diagnosis.—Diseases of the cornea are of the greatest importance, since the acuity of vision depends upon the corneal transparency.

This is more or less impaired in *corneal diseases*, which constitute one-fourth of eye diseases. An early recognition, therefore, of corneal diseases, as well as those of the conjunctiva which implicate the cornea, is imperative in order that by proper treatment the damage to vision be reduced to a minimum.

In all forms of **keratitis** the cornea should be carefully examined with a view of determining the presence of scars, opacities, irregularities of the surface, and the formation of superficial or deep-seated vessels.

Dense opacities are easily recognized, but often lateral illumination is necessary to demonstrate the existence of others.

When there is **loss of substance** involving the corneal surface, an irregularity results which can be recognized by noting the reflection of the window before which the patient should be seated. When the cornea is normal, the picture of the window will be perfect in all positions of the eye; if any irregularity exists, the reflected image of the window will be broken or imperfect.

Superficial vessels occur in **pannus**; deep vessels, in **parenchymatous keratitis**; accordingly the diagnosis of superficial and deep forms of keratitis is facilitated by noting the situation of the vessels.

Complications of Corneal Disease.—Photophobia, blepharospasm, lacrimation, and diminished visual acuity, intra-ocular tension, or sensibility of the cornea may be observed.

Corneal Ulcer.—Etiology.—A corneal ulcer is the result of an infiltrate which, becoming infected, causes an exfoliation of the superficial epithelium over the site of the inflammation and a destruction of more or less of the corneal tissue according to the size and depth of the invaded part.

When the ulcer shows evidence of loss of tissue within an uneven opaque infiltrate, it is in the *progressive stage*, which is characterized by pain, lacrimation, photophobia, and ciliary congestion.

When the formation of the infiltrate stops and the infected tissue is cast off, the ulcer is in the *regressive stage*, in which the signs of irritation diminish or cease in proportion to the completeness of the exfoliation. Bloodvessels from the limbus of the cornea develop to the edge of the ulcer and the regeneration of the tissue begins. This scar-tissue, because of its irregularity of arrangement and compactness, is not transparent, and constitutes the *opacity*.

When the destructive process continues through the cornea, a *perforation* results and the aqueous trickles out, the intra-ocular tension being reduced and the corneal nutrition increased.

The consequences of a perforation are sometimes very serious, since prolapse of the iris, anterior synechia, fistula, anterior polar cataract, dislocation of the lens, intra-ocular hemorrhage, and infection, resulting in an iridocyclitis or panophthalmitis, may occur.

Prognosis.—Opacities through the entire thickness of the cornea remain forever. The patient's age and depth of the ulcer modify the prognosis of corneal opacities. The younger the individual, and the less the corneal tissue involved, the better is the prognosis.

A corneal ulcer is said to be *primary* when it occurs in consequence of some corneal disease; and *secondary* when it is the result of some conjunctival disease.

Treatment.—The treatment of corneal ulcers depends upon the cause; but since there is always infection at the bottom of every ulcer, cleanliness is of importance.

Diseases of the conjunctiva, as trachoma and blennorrhea neonatorum or adultorum, should receive their proper treatment when the ulcer is secondary to these affections. Should *perforation* of the cornea occur, treatment should be continued, with extra care not to injure the cornea by contact with any caustic employed. Copper and lead should not be used in the treatment of conjunctival disease when the cornea is secondarily affected. The bandage is useful in the treatment of corneal ulcers except when the secretions are considerable. Atropine is also of great service. When the ulcer appears rapidly progressive, the application of moist heat, antiseptics, the cautery (electro- or actual), pure carbolic acid, or paracentesis must be resorted to. In some cases autogenous vaccines or serums seem of value. In serpiginous ulcers the pneumococcus serum has been used with benefit.

The serious results of a *prolapsed iris* complicating a perforated cornea should be managed according to the size of the perforation and the duration of the prolapse.

In a favorable case of small perforation there is not any adhesion of the iris to the cornea (anterior synechia).

In other cases a few shreds of the iris remain forever adherent.

A large perforation, allowing prolapse of the iris, is best treated in a recent case by excision of the prolapsed iris in such a way that none of it remains in the corneal opening. This is accomplished by releasing the iris from the edges of the cornea, and after pulling the prolapsed iris well out of the perforation, by cutting it off.

A *corneal fistula* is treated by rest of the patient on the back, the bandage and employment of eserine; but in case of failure, by freshening the corneal edges with a knife or galvanocautery.

When a corneal ulcer is in the stage of *regeneration,* the use of the yellow oxide of mercury salve (gr. viij to the ounce) in a measure reduces the opacity, but the use of dionin in from a 5 to 14 per cent. aqueous solution is the best treatment. The dionin produces considerable reaction immediately after the first instillations, but a tolerance is soon established.

Corneal Abscess.—Etiology and Pathology.—A *corneal abscess* forms in the substance of the cornea, usually in the central part. At the beginning it is surrounded by healthy corneal tissue, of which the overlying portion is slightly depressed. Its margin is the more opaque part. An abscess is due to an abrasion of the epithelial layer of the cornea and the infection of the wound by microörganisms, which may be present in the secretions or in foreign bodies. When the pus gravitates between the corneal layers to the lower corneal margin, it constitutes an *onyx.* When the abscess communicates with the anterior chamber, a *hypopyon,* or accumulation of pus in the anterior chamber, results. When the overlying corneal tissue breaks down, an *ulcer* is formed. An abscess is a very serious disease, since its most favorable termination is a central opacity which is permanent.

In other cases, posterior synechiæ, obliteration of the pupil, or panophthalmitis may result.

Symptoms.—The symptoms of corneal abscess are generally severe: photophobia, ciliary injection, edema of the lids, and pain. An *iritis* always complicates an abscess and posterior synechiæ often occur.

Treatment.—The treatment of corneal abscess depends upon the cause and severity. In simple cases atropine, the bandage, and moist heat suffice. In all cases the disease producing the infective secretion should receive attention. In severe cases cauterization with pure carbolic acid or the electrocautery is useful; and a paracentesis frequently repeated relieves tension and has a favorable influence. After inflammatory signs have subsided, an *iridectomy* is often indicated to make an artificial pupil.

Keratitis.—Special Varieties.—Keratitis e lagophthalmo is due to want of proper protection and lubrication of the cornea. The insufficient secretions evaporate, leaving the corneal epithelium dry, and after exfoliation, infection of the cornea, producing an ulcer or abscess, results. The termination of such a keratitis is that of ulcer or abscess.

The *treatment* of the condition producing the lagophthalmos is of primary importance. In light cases a temporary bandage should be used to protect the eyes; in others a tarsorrhaphy is necessary.

Keratitis neuroparalytica is due to paralysis of the trifacial nerve. In spite of *treatment,* which consists in the use of atropine, bandage, and moist heat, in consequence of the corneal opacity, the sight is lost.

Keratomalacia is a disease of the cornea affecting children, and because of faulty nutrition of the patient the cornea undergoes degenerative changes resulting, if the life is not lost, in opacities of the cornea.

The *treatment* consists in measures to improve the general condition, and the local use of moist heat and the bandage.

Pannus may be a complication of trachoma or phlyctenular conjunctivitis. When due to trachoma, the upper part of the cornea is the usual site, possibly because of irritation of the winking upper lid; but when it accompanies phlyctenular disease, any part of the cornea may become involved.

In the course of *treatment* for phlyctenular conjunctivitis, the pannus, when present, almost entirely disappears, differing in this respect from the pannus of trachoma.

Vesicles and **blebs** occur on the cornea, and damage to the cornea depends upon the cause. Vesicles which accompany febrile diseases leave no bad consequences, as a rule, when proper attention to cleanliness has been observed. Should an ulcer occur, atropine, moist heat, and the bandage are indicated.

Vesicles are sometimes seen on the cornea, complicating herpes zoster ophthalmicus. The *treatment* is the same as above, but the consequences are likely to be more serious, since ulceration more frequently occurs.

Vesicles and bullæ sometimes develop in the cornea of eyes useless from large opacities, iridocyclitis, or glaucoma. The interference with the lymphatic circulation produces edema of the cornea, and vesicles or blebs, according to the size of the elevation, occur. The *treatment* is not very satisfactory, since frequent recurrence of the condition is the rule. When the eye is sightless, an *enucleation* is often the best treatment.

Parenchymatous Keratitis.—Parenchymatous or *interstitial keratitis* (Fig. 18) is a chronic inflammation of the deeper layers of the cornea, usually due to hereditary syphilis, occurring generally between the ages of six and twenty.

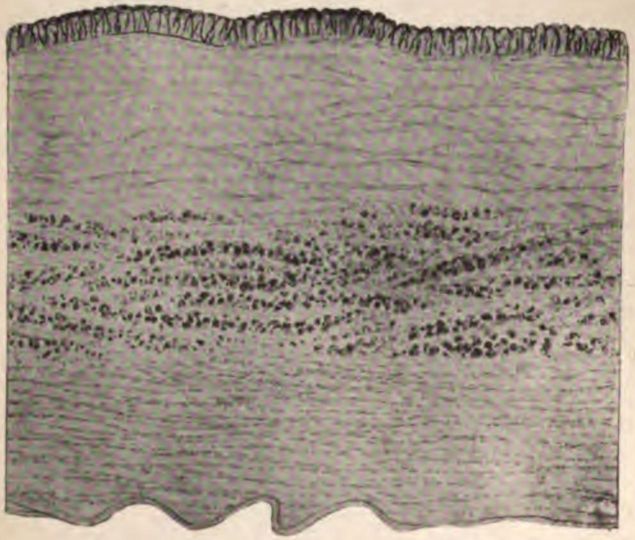

Fig. 18.—Section of interstitial keratitis. (Wedl.)

Etiology.—The etiology is sometimes obscure; may be scrofula, but is in most cases hereditary syphilis. Acquired syphilis very seldom causes primary disease of the cornea.

In many cases evidences of syphilis are striking, one or more of the following being present: a protruding frontal bone, concave nose, poorly developed upper jaw, scars at the angles of the mouth, incisor teeth with concave biting edges, enlarged lymphatic glands, impairment of hearing, and thickening of the periosteum of the long bones, particularly the tibia.

Symptoms.—The clinical picture of the disease varies with the intensity and the place of origin, whether it be at the margin or at the centre of the cornea. The disease usually extends from the margin to the centre of the cornea, in which case the opacity, which is in the deeper layers of the cornea, gradually progresses until the entire cornea presents a milky appearance. The network of bloodvessels which supply the cornea send out branches into the substantia propria. These vessels may take in a large part of the cornea and give it a dull reddish hue. They can be differentiated from the bloodvessels of pannus by the fact that they stop suddenly at the limbus, they have a brush-like appearance, are not distinct, and the surface of the cornea is even, but without luster.

Parenchymatous keratitis beginning at the centre of the cornea is usually more severe. Several whitish opaque spots are seen in the deeper layers of the cornea, and these finally coalesce. The vision is necessarily very much impaired, owing to the central location of the opacity, and the pain, congestion and photophobia are extreme. It will be found difficult at times to dilate the pupil with an atropine solution, but when successful the torn vessels of the iris cause some hemorrhage into the anterior chamber. In all cases, however slight, there is some involvement of the uveal tract, from hyperemia of the iris to a plastic iridocyclitis, which may terminate in atrophy of the bulb.

Treatment.—During the inflammatory stage of keratitis parenchymatosa, atropine, moist heat, and protection from light are indicated. After subsidence of the inflammation the use of some stimulating salve, as the yellow oxide of mercury, or a 5 per cent. to 10 per cent. solution of dionin in water, is of use to dissipate the corneal opacity. The constitutional treatment is also very important, and syphilis, which is usually the cause of the affection, should be treated with mercurials and iodides. In particularly intractable and apparently desperate cases, salvarsan or neosalvarsan should be used when there is no doubt of specificity. The nourishment of the patient should receive attention, and with this end in view the food should be simple and nutritious. Milk and eggs are of great value in these cases; and when the excretions are normal, large amounts of milk are very potent. At times enormous doses of the iodides are not only tolerated, but their favorable influence is manifested by rapid amelioration of the symptoms.

Keratitis Parenchymatosa Circumscripta.—Keratitis parenchymatosa circumscripta is an inflammation involving the middle and deeper layers of the cornea. It differs from the parenchymatous keratitis in that it is circumscribed, the formation of new vessels is slight if there be any, the uveal tract is but slightly involved if at all, and the disease affects only adults.

Etiology.—The etiology is generally obscure, but exposure to cold, malaria, or traumatism has a causal effect in some cases.

Treatment.—The *local treatment* is that of any keratitis—atropine, bandages, and moist heat. The *general treatment* is the use of salicylates or quinine when cold or malaria seems to be the cause.

A *keratitis* may be the result of any condition which interferes with the integrity of the endothelial layer of the cornea. The aqueous humor may, by infiltration, cause a dulness of the cornea. Exudates in the anterior chamber, prolapse, cyst or tumor of the iris, and a dislocation of the lens into the anterior chamber are conditions which may lead to keratitis from absorption of the endothelium.

DEFECTS OF THE CORNEA.

Opacities.—Opacities of the cornea may be due to corneal inflammations, old age, or intra-ocular diseases.

Opacities due to suppurative or non-suppurative corneal disease are classified according to their density as *maculæ* when faint, and *leucomæ* when dense.

When the surface of the cornea is depressed over a small scar from insufficient tissue-regenerations, a so-called *facette* results.

When a large part of the corneal surface is flattened in consequence of a large scar, the condition is known as *aplanatic cornea.*

In old age an opaque semicircle develops near the sclero-corneal margin of the upper half of the cornea, and is soon followed by a similar line in the lower half. These often unite, forming a complete circle, and the segment is known as *arcus senilis* or *gerontoxon.*

In consequence of glaucoma or an iridocyclitis, corneal opacities may also occur; but these are not very significant, since they occur in sightless eyes.

The *treatment* of opacities consists in the use of irritants and the stenopaic spectacles, the correction of the refractive errors, and an iridectomy to establish an artificial pupil, as the case may demand. Opacities, which by their whiteness are disfiguring, may be tattooed with India ink. Only old firm scars occurring in eyes not attacked by iridocyclitis should be tattooed.

Ectasia.—An ectasia (or bulging) of the cornea may be the result of some inflammatory corneal disease; or may occur without any antecedent corneal affection.

Staphyloma and Keratektasia.—Staphyloma and keratektasia are usually the result of ulcerative keratitis. A *staphyloma* is due to a perforation of the cornea in which the iris becomes incarcerated, forming a part of the scar. A *keratektasia* is due to an ulceration which never perforates the cornea, but causes material thinning of it at the site of the bulging. The corneal surface over the keratektasia is always opaque, and thus differs from keratoconus and keratoglobus, both of which present ectasias without antecedent disease.

Treatment.—The treatment of staphyloma, if partial, aims to prevent its progress, and this can at times be effected by paracentesis frequently repeated and a pressure-bandage; or an iridectomy with firm pressure. When complete, an incision through the protruding cornea should be made and a part of the edges excised, in order that the cicatrix may become more flattened. Enucleation is at times necessary to relieve pain and prevent sympathetic inflammation.

Keratektasia is not amenable to treatment.

Keratoconus.—Keratoconus is a rare disease, the cause of which is not understood. It is characterized by a thinning of the central part of the cornea and a conical projection.

Treatment.—Tonics and myotics have had some influence in checking the disease. Cauterization or excision of the apex and an artificial pupil by performing an iridectomy have been successful in a few cases.

Keratoglobus.—Keratoglobus is a feature of hydrophthalmos in which the cornea is larger and more bulging than normal.

THE IRIS.

ANATOMY.

The choroid, ciliary body, and iris form the **uveal tract.**

The ciliary body, or middle zone, connects the choroid, or posterior zone, and the iris, or anterior zone.

The iris arises from the anterior surface of the ciliary body, being reinforced by some fibers of the ligamentum pectinatum iridis and the inner wall of Schlemm's canal.

The **pupil** is the central perforation of the iris, and is subject to variations of size. The iris divides the aqueous chamber, the space in front of the lens and suspensory ligament into an anterior and a posterior chamber, between which there is normally free communication through the pupil.

The **anterior surface** of the iris is divided by an uneven circular line into two zones, a pupillary and a ciliary. Near the circular line in the ciliary zone are numerous *facets;* nearer the periphery are several *rings* or furrows concentrically arranged.

The *lesser arterial circle* of the iris produces the uneven circular line, and the *radiating lines* extending from the periphery to the pupil represent small vessels in the vascular layer.

The **iris consists** of an anterior endothelial layer, stroma, or vascular layer, a muscular layer, and a pigment layer.

The **anterior endothelial layer** is a continuation of a part of

Descemet's membrane, and is wanting over the facets of the ciliary zone.

The **stroma**, or **vascular layer**, contains the arteries and veins, between which are round and branched cells held in place by connective-tissue fibers.

The two *long posterior ciliary* arteries supply the iris, forming two arterial circles; the peripheral one being the circulus arteriosus iridis major, the radiating branches of which near the pupil form the second circle, circulus arteriosus iridis minor.

The amount of **pigment** in the cells of the stroma determines the color of the iris, except in the case of albinism, when there is no pigment present anywhere in the iris. The iris appears blue when there is no pigment in the stroma, gray when there is a slight amount, and brown when there is very much.

The **muscular layer** contains two kinds of fibers, one constituting the *sphincter pupillæ*, the other the *dilator pupillæ*. These fibers lie in the posterior part of the vascular layer.

The *sphincter pupillæ* is supplied by the motor oculi, of which direct or reflex stimulation produces contraction of the pupil. Paralysis of the third nerve causes dilatation of the pupil.

A branch of the cervical sympathetic to the lenticular ganglion supplies the *dilator pupillæ*. Stimulation of this nerve causes dilatation, paralysis, contraction of the pupil.

Not only the sphincter of the iris, but the ciliary muscle and the internal recti are supplied by the third nerve, and since the centres of accommodation, pupillary reaction, and convergence are intimately connected in the nucleus of the motor oculi in the floor of the Sylvian aqueduct, normal convergence and accommodation are accompanied by a contracted pupil.

This phenomenon is known as the *associated reaction* of the pupil.

Moreover, when light stimulates the retina of one eye (the other being covered), causing a contraction of the pupil, a similar contraction of the pupil of the covered eye also occurs. This is known as *consensual reaction*, and is due to the fact that the stimulus of one eye, owing to the decussation of the optic nerve fibers in the chiasm, passes along both optic tracts to the nuclear centres in the floor of the Sylvian aqueduct and back again by way of the third nerve to both irides.

The *Argyll-Robertson* pupil is one which does not react to light, whereas convergence and accommodation cause reaction, and is due to a change in the fibers of the optic tract which go to the floor of the Sylvian aqueduct (Meyners's fibers) or in the portion of the nucleus of the third nerve presiding over the light reflex.

CONGENITAL ABNORMALITIES OF THE IRIS.

The posterior epithelial layer and the cells of the vascular layer, or stroma, contain the *pigment* which determines the color of the iris. When there is no pigment in the posterior epithelial layer, it is always absent in the stroma.

This condition occurs in *albinism*. The iris of albinos does not sufficiently exclude an excess of luminous rays, and hence these people half-close their lids in bright light. The vision is poor, and nystagmus frequently complicates the trouble.

Corectopia.—Corectopia is the condition in which the pupil in one or both eyes is more or less eccentric. Slight alterations of position frequently occur; extreme cases are rare, and there are usually complications of dislocated lenses.

Microcoria.—Microcoria is a small pupil, and is generally displaced. It may be due to adhesions of the iris to the lens (posterior synechiæ) occurring in consequence of a fetal iritis.

Discoria.—Discoria is a pupil abnormal in shape without a coloboma of the iris. It may be due to posterior synechiæ or persistence of a part of the pupillary membrane.

Polycoria.—Polycoria is that condition in which there are two or more openings of the iris. As many as nine openings have been observed.

Irideremia.—Irideremia, or *aniridia*, is the term applied to the condition in which there is an absence of the iris. It may be partial or complete, but is a bilateral abnormality. Aniridia is possibly due to a late separation of the lens and cornea, the entire anterior surface of the lens being involved.

On ophthalmoscopic examination, a large illuminated area representing the size of the lens is observed, and at times the fibers of the zonula can also be seen. By partially closing the lids some light is excluded, and this is the method the patient uses to exclude an excess of light. The vision is poor and nystagmus is often present.

Coloboma.—Coloboma of the iris is the condition in which a part of the iris is deficient, causing an abnormality in the size and shape of the pupil. As the iris is a prolongation of the ciliary body, clefts in the choroid and ciliary body interfere with the development of the iris.

A coloboma might also be due to an abnormal adhesion between the lens and the cornea, occurring during the embryonal stage, the defect of the iris representing the area of adhesion. An uncomplicated coloboma of the iris does not materially prejudice vision; when, however, a coloboma of the choroid, ciliary body, or lens is present, vision is more or less impaired.

Iridodonesis.—Iridodonesis is the term that expresses a tremor of the iris. In buphthalmos, or in eyes with an absence or dislocation of the lens, the iris has not its normal support and is tremulous.

The **pupillary membrane,** which begins to disappear between the seventh and eighth months of fetal life, is that part of the anterior fibrovascular sheath extending across the pupil. The anterior fibrovascular sheath consists of a meshwork of bloodvessels coming from the capsule of the lens: the periphery forms the anterior layer of the iris; the central portion, the pupillary membrane. As the eye develops the hyaloid artery becomes smaller, and as the nutrition of the lens from this source is diminished the fibrovascular sheath disappears.

When for any reason a part of the sheath over the pupil does not disappear, the condition known as *persistent pupillary membrane* results. This abnormality is one of the most frequent of the congenital anomalies of the eye. It can be distinguished from posterior synechiæ by the fact that the pupillary membrane arises from the anterior surface of the iris.

NEOPLASMS OF THE IRIS.

Cystic Tumors.—Cystic tumors of the iris may contain a serous liquid or atheromatous material. They are very rare, and develop usually after operations whereby epithelium from the skin, conjunctiva, or cornea is carried into the wound. These cysts grow very slowly, and at the beginning may be removed by performing an iridectomy of the part involved. If left alone, they may obstruct the pupil, displace and opacify the lens, cause iritis and iridocyclitis, and, by increase of intra-ocular tension (secondary glaucoma), destroy the eye.

Sarcomas.—Sarcomas of the iris may be either pigmented or non-pigmented. They gradually fill the anterior chamber and finally perforate the eyeball. When very small, the excision of the involved part of the iris may be sufficient, but enucleation is the safest treatment.

Tuberculosis.—Tuberculosis of the iris is a rare disease more often affecting tuberculous children. The tubercles may be disseminated or solitary.

Attention to the general health and measures to relieve the iritis constitute the *treatment* in favorable cases. Tuberculin should be used for months if necessary, but in hopeless cases enucleation is indicated to remove the source of infection.

INJURIES OF THE IRIS.

Effects.—Injuries whereby the iris is torn or lacerated are likely to produce *iritis*. When infection is carried into the eye through a wound involving the ciliary region or iris, and *iridocyclitis* is the result. *Simple contusion* of the bulb may cause only a *transient hyperemia* of the iris; but *penetrating wounds* when *infected* may cause not only the *loss of sight* of the injured eye, but also *sympathetic inflammation* of the other.

Prognosis.—The prognosis of traumatic iritis depends upon the site of injury (the ciliary region being the most dangerous), the cleanliness of the wound, and the presence or not of foreign bodies in the eye.

In such conditions as are likely to produce sympathetic inflammation, the eye should be *enucleated*.

Iridoplegia and Cycloplegia.—Iridoplegia and cycloplegia are terms designating respectively paralysis of the sphincter iridis and ciliary muscle. These conditions may occur in consequence of *contusion* of the eye, and from this cause are generally permanent. *Hyphemia* (blood in the anterior chamber) is usually observed in such cases.

Treatment.—The treatment consists in rest and application of cold at the beginning, and later instillation of eserine if not contra-indicated by an iritis.

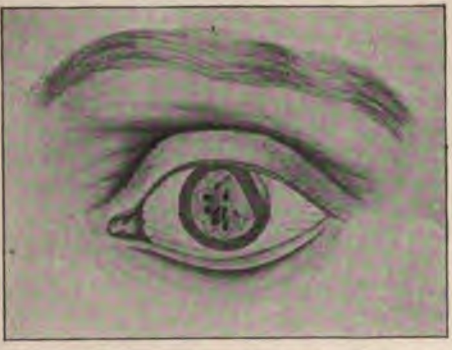

FIG. 19.—Traumatic iridodialysis. (Jäger.)

Iridodialysis (Fig. 19).—Iridodialysis is the term that expresses a detachment of the iris from the ciliary body. The detachment varies in extent; it may be very small or the entire iris may be torn from the ciliary attachment. In partial detachment monocular diplopia may occur when two pictures are projected upon the retina, the one through the pupil, the other through the gap. Total detachment of the iris is called an *aniridia traumatica;* loss of a part of the iris, *coloboma traumaticum*. In all cases of iridodialysis hyphemia is present, owing to rupture of the vessels of the iris.

Treatment.—The tendency to hemorrhage indicates the use of cold and observance of quiet. If *iritis* develops, atropine must be used.

In consequence of *penetrating injuries* of the eyeball a *cyst* in the iris-angle, due to an accumulation of aqueous in an inversion of the pectinate ligament and adjoining endothelium is occasionally observed.

It should be removed before it occasions cyclitic irritation or increase of intra-ocular tension. A cyst may also occur in the *parenchyma* of the iris from like injury, and can be removed with the portion of the iris in which it lays.

Foreign Bodies.—Foreign bodies in the iris are rare, but chips of metal or stone have been known to become encysted and remain for years. Foreign bodies should be removed even if an iridectomy be necessary.

DISEASES OF THE IRIS.

Iritis.—**Definition.**—Iritis is an *inflammation of the iris* characterized by severe neuralgic pain about the orbit, photophobia, lacrimation, engorgement of the ciliary vessels, muddy appearance of the iris, sluggishness of the pupil, and more or less disturbance of vision.

Etiology.—Iritis may be primary or secondary.

Primary iritis is sometimes caused by constitutional diseases, as syphilis, scrofula, tuberculosis, rheumatism, gonorrheal rheumatism, acute infectious diseases, or diabetes. Traumatism and sympathetic inflammation are local etiological factors. An idiopathic iritis of which the cause is probably bacterial may also occur.

Secondary iritis frequently complicates corneal diseases and scleritis; retinal detachment and choroiditis less often cause it.

Symptoms.—The symptoms of iritis are pain, photophobia, lacrimation, conjunctival congestion, circumcorneal or ciliary redness, imperfect action of the pupil, exudation of serous, plastic, or purulent material into the parenchyma of the iris, upon its anterior or posterior surface, into the pupillary area or the aqueous, and impaired vision. One or more of these symptoms may be present, so that the diagnosis is usually not difficult. The use of atropine is an invaluable *diagnostic agent* in some cases: the slow and imperfect response of the iris to the mydriatic influence of the alkaloid and the irregularity of the pupil are pathognomonic.

An iritis may occur alone, but it at times involves the *ciliary body,* producing an iridocyclitis; in which case there may be present, besides the ordinary symptoms of an iritis, edema of the lids, pain on pressure over the ciliary region, complete posterior synechiæ, impairment of vision, all out of proportion to the condition of the anterior chamber, or change of tension.

Differential Diagnosis.—Iritis is frequently mistaken for *conjunctivitis;* and sometimes an *acute inflammatory glaucoma* is thought to be an iritis. Both mistakes are serious, and the latter may be disastrous.

In *conjunctivitis,* however slight, there is more or less mucous discharge, which glues the lids together. In *iritis,* however severe, the secretions are thin and the lids are never glued together. The use of atropine soon shows the existence of an iritis, in which case the pupil either does not dilate at all or yields slowly and imperfectly. When *posterior synechiæ* have formed, the pupil is irregular in shape.

In *iritis* the pupil is usually small; in *glaucoma,* large. In *glaucoma,* moreover, the tension is increased; there are anesthesia of the cornea and a halo around lights. The use of atropine in glaucoma might destroy sight in a few days by increasing the intra-ocular tension. Hence the differential diagnosis between glaucoma and iritis becomes a very important consideration in the use of atropine.

Prognosis.—The prognosis of an iridocyclitis is more serious than that of a simple iritis, as might be inferred from the severity of the symptoms.

Consequences.—The consequences of an iritis or iridocyclitis which impair the usefulness of the eye are *exudates* and *synechiæ.*

Posterior synechiæ when small occasion little or no damage to signt, but when circular (seclusio pupillæ) the drainage of the nutrient liquid supplied by the ciliary processes from the posterior to the anterior chamber is interfered with, and the accumulation of the liquid in the posterior chamber causes a bulging and finally atrophic iris; and by increase of intra-ocular tension a secondary glaucoma results.

When the pupillary area is filled with *exudate* the sight is materially impaired. When an exudate imbeds the lens, from faulty nutrition, a cataract results; when the exudate displaces the vitreous, shrinking of the bulb follows, and sight is lost.

Treatment.—The treatment of iritis and iridocyclitis is constitutional, local, and surgical, according to the cause and complications.

The appropriate constitutional treatment should be instituted when syphilis, rheumatism, malaria, diabetes, gout, anemia, scrofula, tuberculosis, or gonorrhea seems to be a causal factor of the iritis.

Atropine should be employed in all cases, unless contraindicated by increase of tension, to dilate the pupil until the inflammation has subsided. The atropine paralyzes the ciliary muscle and by dilating the pupil prevents the formation of adhesions to the lens and reduces the congestion of the iris and exudation. Light adhesions to the lens are frequently disturbed by timely use of atropine.

Pain and inflammation are reduced by the application of heat, either moist or dry, or of cold over the eye. The feelings of the patient are of importance in the selection of heat or cold, but heat will be generally found the more acceptable. The abstraction of blood from the temples by leeches (three or four will suffice) or by the artificial leech sometimes gives immediate relief; but this should only be practised in plethoric subjects.

Narcotics and the coal-tar preparations are sometimes indispensable in affording relief from pain.

When the signs of sympathetic iritis are slight, enucleation of the injured eye may save the other; but when the symptoms are well developed, no treatment is of use.

Paracentesis and iridectomy are the surgical procedures advocated for the relief of persistent increase of tension occurring during the acute stage of iritis or iridocyclitis. Paracentesis should first be tried, and if unsuccessful, an iridectomy may be thought of when the iris has not undergone great structural changes.

The *consequences* of iritis and iridocyclitis which call for treatment are small posterior synechiæ, the evidences of which are notching of the pupil; annular or circular posterior synechiæ (exclusio pupillæ); and total posterior synechiæ, in which the entire posterior chamber is filled with exudate. Small localized posterior synechiæ often yield to atropine and cocaine in combination. Eserine, followed by atropine, whereby the pupil is dilated after myosis has been produced, is a successful method in the smaller synechiæ, but more extensive adhesions resist all but operative treatment. An annular posterior synechia calls for operation as soon as the eye becomes quiescent, in order to restore the communication between the posterior and anterior chambers. In a simple case the iridectomy should be made in the upper segment; but when the pupil is occluded the lower nasal side should be selected in order to favor vision. A total posterior synechia requires an iridectomy, but since it is not generally possible to remove a piece of the iris sufficiently large, extraction of the lens will often be necessary. An iridectomy may be done when the lens is shrunken or absent.

ABNORMALITIES OF MOVEMENT OF THE IRIS

These include *mydriasis, myosis, anisocoria, iridodonesis,* and *hippus.*

Mydriasis.—Mydriasis, or dilatation of the pupil, may be spasmodic or paralytic. When spasmodic, it may be a symptom of cerebral or spinal disease, or due to irritation direct or reflex of the cervical sympathetic.

Paralytic mydriasis is much more common, and is due to paresis or paralysis of the motor oculi; cerebral diseases affecting the nucleus of the third nerve; use of mydriatics, as atropine, homatropine, daturine, and scopolamine; poisoning by ptomaines or the products of the diphtheria bacillus; syphilis; pressure on the ciliary nerves from increase of intra-ocular tension; and injury producing iridoplegia.

The *prognosis* and *treatment* depend upon the cause. Mydriasis from injury, cerebral diseases, and glaucoma is often permanent.

Myosis.—Myosis, like mydriasis, may be spasmodic or paralytic. When spasmodic, the myosis is due to agents like eserine and pilocarpine; when paralytic, to paralysis of the cervical sympathetic and to spinal disease, as tabes.

Anisocoria.—Anisocoria is a term which designates a difference in the size of the pupils. In young nervous individuals, mydriasis, seldom complete, may occur, but the condition is temporary, although subject to relapses. Syphilis is the more common cause of unilateral mydriasis, and yields to treatment.

Cycloplegia, or paralysis of the accommodation, sometimes complicates the condition.

Iridodonesis.—Iridodonesis, or pathological trembling of the iris, occurs when the iris, because of the absence of the lens, has not the proper support.

Hippus.—Hippus is a condition in which the size of the pupil is subject to rapid variations because of clonic or intermittent spasm of the iris. It occurs in various nervous diseases, both organic and functional.

THE CILIARY BODY.

ANATOMY.

The **ciliary body** connects the choroid and the iris, forming the middle zone of the uveal tract (Fig. 20).

It consists of the *ciliary muscle,* composed of unstriated muscle fibers, *connective tissue, bloodvessels,* and *nerves;* and the *ciliary processes,* which are similar in structure to the choroid, with the exception that the arteries empty directly into the veins without the intervention of capillaries.

The *inner surface* of the ciliary body is a continuation of the pigment-layer of the retina, and is known as the **pars ciliaris retinæ.**

Anteroposteriorly the ciliary body is about one-quarter of an inch in width, and a vertical section of the bulb shows that it is triangular in shape, the base being directed forward and the apex merging into the choroid.

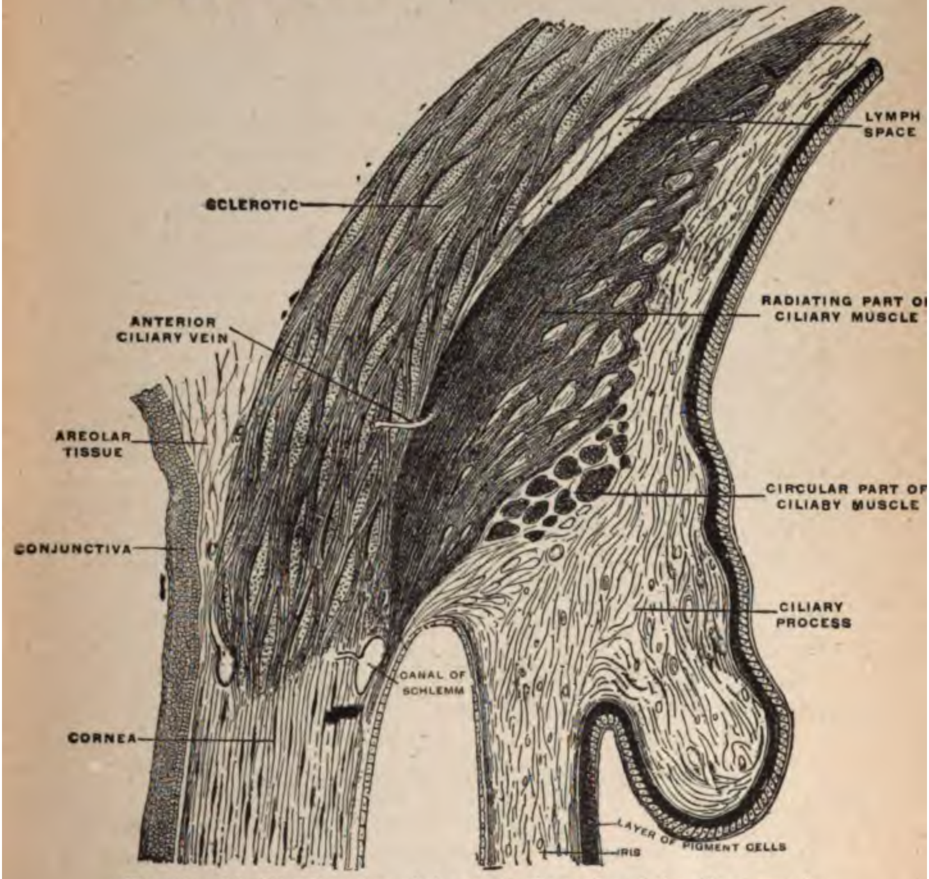

Fig. 20.—Meridional section of the eye in the region of the ciliary muscle. (Testut.)

The **ciliary muscle** consists of longitudinal, radiating, and circular fibers, arranged in this order from without inward. The **ciliary processes** represent the choroid and the pigment-layer of the retina: the former is external, the latter internal.

The processes anteriorly are thrown into from seventy to eighty folds, the posterior portion is perfectly smooth.

The *ciliary muscle* is the means of effecting *accommodation* for vision at different distances. When the eye is at rest, implying rest of the ciliary muscle, the lens is flattened by the tension of the suspensory ligament, which is produced by the position of the ciliary body. When the ciliary muscle is at rest, the ciliary body is at a greater distance from the equator of the lens than during accommodation, and the anterior border of the ciliary body during rest extends farther backward. The position of the ciliary body being such that the tension of the suspensory ligament is increased during rest of the ciliary muscle, it will be evident that the lens is flattened from the tension of the suspensory ligament when the eye is at rest. When the tension is removed because of the change of position of the ciliary body, which is nearer the lens during accommodation, the natural elasticity of the lens increases its convexity. During accommodation the entire iris moves forward and the pupil is contracted.

The *ciliary processes* supply nutrition to the lens and contiguous portion of the vitreous, and replenish largely the loss of the aqueous humor.

The ciliary muscle is *supplied* by fibers from the third nerve, which also supplies the sphincter pupillæ and internal recti muscles. The intimate connection of these fibers explains the convergence of the eyeball and contraction of the pupil which are associated with accommodation.

ANOMALIES AND NEOPLASMS.

Coloboma.—Coloboma of the ciliary body is due to a persistence of the cleft, and is often associated with a coloboma of the iris, choroid, or lens. Inasmuch as the iris grows from the anterior border of the ciliary body, a coloboma of the ciliary body is the cause of the coexisting coloboma of the iris.

Neoplasms.—Newgrowths, excluding tubercle and gummata, are very rare. When present, enucleation is the *treatment*.

INJURIES OF THE CILIARY BODY.

Wounds.—Wounds of all kinds may involve the ciliary region, and, because of the danger to the uninjured eye from sympathetic inflammation, are of the greatest importance.

Injuries of the choroid or iris from a *foreign body* are very serious from the possibility of causing sympathetic trouble. But if the ciliary region be involved the danger is the greatest. The nearer the wound is to the ciliary region, the greater becomes the danger.

A *sympathetic inflammation* is usually the result of trauma affecting the iris, ciliary body, or choroid, whereby, after a variable time (from a few weeks to many years), a plastic or serous uveitis occurs in the other eye.

The most popular theory explaining the production of sympathetic inflammation is the one which teaches that infection is conveyed to the other eye along the optic nerve and its sheaths.

When an injured eye some time after receipt of the injury becomes painful and the other presents signs of irritation, such as photophobia, lacrimation, congestion, or tenderness, sympathetic inflammation should be thought of. Usually it will be noticed that the accommodation of the uninjured eye is crippled. The ciliary body is involved at first, then the iris, and finally the choroid.

Treatment.—Treatment of sympathetic inflammation is most satisfactory when prophylactic. An injury in the ciliary region, the eye being sightless, is best treated by an enucleation. When a panophthalmitis occurs in consequence of traumatism, the danger of sympathetic inflammation is not great.

When sympathetic inflammation has fairly developed, treatment is useless. If any vision remains in the injured eye after marked signs of sympathetic ophthalmitis, it should not be enucleated, since the sight of the injured eye is then apt to be all the patient will possess. If some time after injury to an eye a patient presents himself with tenderness and painfulness of that eye, it is well to suggest an enucleation as the only treatment.

In consequence of injury, cycloplegia or paralysis of the ciliary muscle often accompanies iridoplegia. Cycloplegia from traumatism is usually permanent.

DISEASES OF THE CILIARY BODY.

Cyclitis.—An inflammation of the ciliary body seldom occurs alone, the iris being almost invariably affected. An iritis may occur without involving the ciliary body, but sometimes the ciliary body becomes secondarily affected. An *iridocyclitis* is an inflammation of both the iris and ciliary body.

A *simple cyclitis* presents many symptoms of an iritis—pain, photophobia, lacrimation, ciliary redness, change of color of the iris, impairment of sight, and evidences of exudate; but besides these, a *hypopyon,* which rapidly appears and disappears, a *deposition of particles* upon the posterior surface of the cornea (constituting keratitis punctata), tenderness over the ciliary region, and increase of intra-ocular tension occur. When the cyclitis is due to sympathetic inflammation, a whitening of the eyebrows is sometimes observed. A simple cyclitis is usually a chronic trouble, and is subject to frequent exacerbations.

The *causes* of cyclitis are the same as those of iritis, and the *treatment* is the same.

THE CHOROID.

ANATOMY.

The *choroid,* ciliary body, and iris form the middle coat of the eye, and are known as the **uveal tract.**

The choroid, forming the posterior seven-tenths of the middle tunic, extends from the entrance of the optic nerve to the ora serrata, where the ciliary body begins. It is loosely attached to the sclera, except at the margin of the disk, where there is a ring of connective-tissue fibers. The choroid is from $\frac{1}{250}$ to $\frac{1}{150}$ of an inch in thickness and consists largely of bloodvessels (Plate III).

The lamina fusca, tunica vasculosa, membrana choriocapillaris, and membrana limitans are the four layers of the choroid.

The **lamina fusca**, or suprachoroidea, consists of loose cellular connective-tissue fibers, among which are imbedded many pigment cells, lymph corpuscles, and elastic fibers. This layer loosely connects the choroid and the sclera, between which is the perichoroidal lymph space, which communicates with the lymph space in Tenon's capsule.

The **tunica vasculosa** contains the ramifications of the long and short posterior ciliary arteries, which are internal to the veins. These arteries empty into the choriocapillary layer, and the blood from this layer and much from the ciliary body and iris leave the eye by the *vorticose veins*, of which there are from four to six. The smaller veins empty into larger ones, which finally form a single large vein, or *vena vorticosa*, which is the summit or vortex of each group or set of veins.

The **membrana choriocapillaris** contains the capillary system formed by the long and short posterior ciliary arteries.

The **membrana limitans**, or *lamina vitrea*, is very thin, and is firmly attached to the membrana capillaris. The pigment layer of the retina covers the inner surface of this layer.

The choroid and ciliary processes furnish **nutritive lymph** to the lens, anterior part of the vitreous, and external layers of the retina.

CONGENITAL ANOMALIES OF THE CHOROID.

Albinism.—In cases of complete albinism, the choroidal and retinal pigment is wanting, and the choroidal vessels can be easily seen upon a light background with the ophthalmoscope. The pupil presents a pinkish reflex. The visual acuity is always diminished, and the eyeballs make rapid oscillations, a condition known as nystagmus. Myopia and strabismus often complicate albinism.

Coloboma.—Absence of the choroid usually occurs in the choroidal fissure, but may occur in the area of the macula.

With the ophthalmoscope an oval whitish area is seen extending from the lower margin of the disk toward the ciliary body, the long diameter being that of the anteroposterior axis of the globe. Because of the absence of the pigment and the choroidal vessels the white sclera is seen, representing the size of the defect. When the retina is perfect, its vessels are seen to cross the area of defect; when absent, the vision is all the worse.

Coloboma in the choroidal fissure is often accompanied by similar defects of the ciliary body and iris, as well as other anomalies of development. A small crescentic defect of choroid at the inferior margin of the disk stimulates the myopic crescent in appearance, but not in location. This is known as the coloboma of Fuchs.

A coloboma of the choroid about the disk may or may not be associated with a coloboma of the nerve sheath.

Colobomas are often hereditary, and occur in individuals having other congenital malformations.

The size and position of the coloboma and the condition of the eye determine the impairment of vision.

Treatment is useless.

NEOPLASMS OF THE CHOROID.

Sarcoma.—Sarcoma, usually a melanosarcoma, is a very rare growth of the choroid, occurring most frequently between the ages of forty and sixty. Since *glioma* of the retina occurs exclusively in children, the patient's age is a diagnostic point of value, inasmuch as both neoplasms run a similar course.

Symptoms.—The *first stage* of the growth can be diagnosed only with the ophthalmoscope, more or less detachment of the retina and corresponding disturbance of vision being present.

The *second stage* is that in which a secondary glaucoma or, more rarely, an iridocyclitis results.

The *third stage* is characterized by perforation of the bulb, and the *fourth* by the occurrence of metastatic nodules in other organs.

Prognosis.—The prognosis is bad as regards life, for most patients succumb unless the malignant neoplasm be removed at the beginning.

Treatment.—At times enucleation does not suffice, and an exenteration of the orbit, including the periosteum, is necessary.

INJURIES OF THE CHOROID.

Contusion.—Contusion may produce a *rupture* of the choroid, which is either a simple condition or a complication of other injuries from the same cause. Immediately after the injury the diagnosis may be difficult, owing to the hemorrhage.

The rupture usually appears like a rent or streak, which is sometimes forked and is often concentric with the optic papilla. Unless the rupture has involved the retina, the retinal vessels will be seen to pass over the rent. The location of the rupture determines the impairment of vision, the macular region being the worst.

Treatment.—Treatment consists in rest, atropine, and antiphlogistic measures, as use of cold cloths and local blood-letting.

Detachment.—Detachment of the choroid sometimes occurs, but is not recognized, owing to the opacity of the media, until the eye has been enucleated. When the choroid is injured, in consequence of a *penetrating wound*, the condition is very bad, particularly when infection has taken place.

Foreign Bodies.—Foreign bodies which injure the choroid in their course should be removed on general principles.

Injuries producing a *suppurative choroiditis* lead to panophthalmitis and phthisis bulbi, with loss of sight and shape of the eye.

CHOROIDITIS.

The choroid is subject to three kinds of inflammations: tubercular; an exudative or plastic; and a suppurative.

Tuberculosis of the Choroid.—The nodule may be solitary; or multiple and disseminated, When *solitary*, a very rare condition, the course of the disease is that of a neoplasm, and most often occurs in youth. The eye is invariably lost, but the patient's life may be saved by an *enucleation* if the eye is the single and primary seat of the affection.

Disseminated tuberculous nodules, usually less than ten in number, are a manifestation of miliary tuberculosis, and when seen with the ophthalmoscope, assist in diagnosis of the same. The nodules can be differentiated from other inflammatory choroidal changes (choroiditis disseminata) by their rapid growth and absence of pigment.

Exudative Choroiditis.—**Pathology.**—It is manifested by the formation of an exudate at the site of inflammation. Absorption of the exudate leads to atrophy of the choroid and development of pigment, which more often accumulates at the margins of the diseased spots. In some cases the choroid alone is diseased, but frequently the retina is more or less involved, in which case the condition is called a *chorioretinitis*.

An exudative choroiditis does not, as a rule, involve the iris and ciliary body as does the suppurative form, and the media remain transparent, so that the diagnosis with the ophthalmoscope is an easy matter. An exudative choroiditis may affect one or both eyes, and the **cause** is often syphilis, either hereditary or acquired, scrofula, anemia, and diseases which lower the vitality.

According to the location and arrangement of the lesions, the implication of the retina, or the cause, exudative choroiditis is classified as follows: *choroiditis disseminata; choroiditis areolaris; choroiditis centralis; chorioretinitis specifica*, and *staphyloma posticum*, or posterior sclerochoroiditis.

Choroiditis Disseminata.—Choroiditis disseminata is characterized by a number of spots which are scattered about the fundus, the papilla representing the centre. The atrophy of the choroid permits the view of the sclera, so that the spots are yellow or white, according to the degree of atrophy. The disease usually begins at the periphery and extends toward the papilla. Sometimes the lesions are confined to the periphery, the papilla and circumpapillary portion of the choroid showing evidences of atrophy. These cases usually develop in syphilitic subjects. In some old cases of disseminated choroiditis the retina becomes involved, so that the deposition of pigment may cover the retinal vessels, which, because of a diminution in size, indicates an atrophy of the retina. The papilla is often blurred, the margins being indistinct and the cupping not demonstrable. Such cases receive the name *chorioretinitis.*

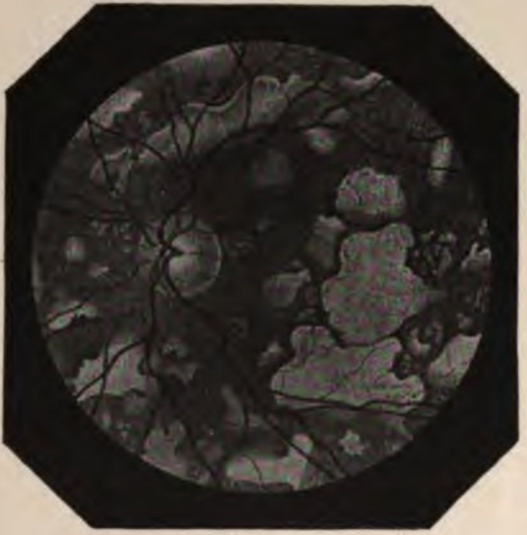

FIG. 21.—Syphilitic choroiditis. (Jäger.)

Choroiditis Areolaris.—Choroiditis areolaris presents for examination lesions of which the macular region forms the centre. The lesions form about the macula in such a way that the recent spots are the more distant.

Choroiditis Centralis.—Choroiditis centralis has a lesion which covers the macular region, producing a central scotoma or blind spot. High degrees of myopia, syphilis, and contusions may produce it.

Chorioretinitis Specifica.—Chorioretinitis specifica is due to syphilis (Fig. 21), and involves the retina and choroid, as the name implies. The vitreous and retina become less transparent, and in the vitreous small dust-like particles develop. A papilloretinitis, characterized by a haziness of the papilla and circumpapillary portion of the retina, is usually present. Atrophy of the retina with its vessels notably diminished in size and a deposition of pigment are the results of this disease.

Posterior Staphyloma.—A posterior staphyloma is that condition of the choroid characterized by an atrophy adjacent to the disk associated with a bulging of the sclerotic, which is greatest at the temporal border of the disk. This condition is the cause of malignant forms of myopia and often complicates myopia of high degree.

Treatment.—The treatment of all forms of exudative choroiditis depends upon the cause. Syphilis and the various dyscrasias demand their particular treatment. To facilitate absorption of the exudates, the iodides and diaphoretics (as pilocarpine and the salicylates) are indicated. Absolute rest of the eye and a darkened room are necessary for some cases.

Suppurative Choroiditis.—**Etiology.**—This disease is due to infection, the source being either within the patient—endogenous—producing the disease by metastasis; or from without—exogenous. Phlegmonous inflammation of the orbit, meningitis, and pyemia are the more common sources of endogenous infection. Traumatism and spontaneous perforations of the cornea in consequence of ulcers or abscesses may produce infection from without.

Symptoms.—This disease is associated with secondary inflammation of the ciliary body and iris, and the symptoms are therefore intense. The lids are swollen, the conjunctiva is congested and edematous, the cornea dull, the aqueous is no longer transparent, and a small amount of pus settles at the bottom of the anterior chamber—hypopyon. The iridocyclitis produces great pain and tenderness. The choroidal exudate which occurs between the choroid and retina extends into the cavity of the vitreous. The intra-ocular pressure is increased because of the exudate and possible interference with drainage, due to a seclusio pupillæ in the first stages. In less severe cases the globe begins to shrink, absorption taking place: *Atrophy;* in severer cases the globe becomes an abscess—panophthalmitis, and the pus finally finds an exit through the sclera, leading to a condition known as *phthisis bulbi.*

Diagnosis:

PHTHISIS BULBI.	ATROPHIA BULBI.
Follows panophthalmitis, and the eyeball is diminished in size from escape of some of the contents through an opening.	Follows iridocyclitis, iridochoroiditis serosa, or suppurative choroiditis (not severe). The eyeball becomes smaller from absorption of exudate.
The contents of the eye are more or less destroyed or lost, the choroid in particular. Sympathetic inflammation is not so likely to occur, and enucleation, therefore, is not often necessary.	The contents of the eye are not necessarily much damaged, and sympathetic inflammation is possible. Enucleation of the eye is frequently called for, to prevent sympathetic inflammation and relieve pain.
Eyeball is considerably reduced in size in a short time.	Eyeball gradually diminishes in size.
Artificial eye may be used.	Artificial eye contra-indicated.

When an *exudate*, the result of choroiditis, can be seen behind the lens in children under two years of age, the condition is named *pseudoglioma,* from the possibility of mistaking it for a glioma.

Treatment.—The treatment of suppurative choroiditis is palliative. To relieve the pain, heat and anodynes are useful. An enucleation, if performed in the early stages, is not dangerous, and much suffering is forestalled. An incision may be made through the sclera and evacuation of the pus thus hastened. In the later stages an enucleation is dangerous, since a suppurative meningitis may result.

GLAUCOMA.

Definition.—Glaucoma is a disease of the eyeball, characterized by an increase of tension.

Varieties.—As regards its duration, it may be *acute, subacute,* or *chronic;* and it is either *primary*, when there has been no antecedent causal disease; or *secondary*, when the increase of tension is due to some previous ocular affection.

Primary glaucoma may occur with or without inflammatory manifestations, and is accordingly called either *inflammatory* or *simple*. *Hydrophthalmos* is a disease of childhood, characterized by an extraordinary size of the eyeball and an increase of tension which is not accompanied by inflammatory reaction: it is therefore a form of simple glaucoma.

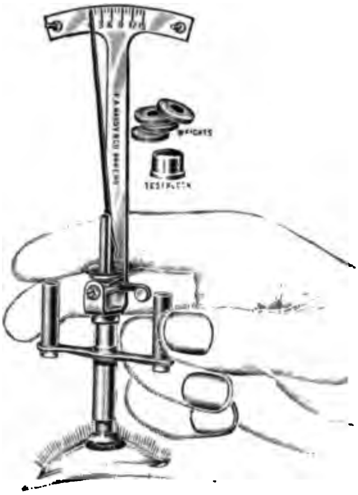

FIG. 22.—Tonometer.

Normal tension of the eyeball is expressed by the letter T; *increased tension* (hypertony), according to the degree, by T + 1, T + 2, or T + 3; *reduced tension* (hypotony), according to the degree, by T – 1, T – 2 or T – 3. T + 1 ? signifies doubtful increase of tension, and T – 1? doubtful reduction of tension. To determine the tension, palpation with the two index fingers, the hands resting on the patient's face, is a ready method. For absolute accuracy, however, the tonometer (Fig. 22) should be employed. The theory of this instrument is based upon the fact that a difference of pressure is necessary to flatten a given area of the cornea, according to the intra-ocular tension. After instillation of a solution of holocaine (0.5 to 1 per cent.) the instrument is placed perpendicularly upon the cornea of the patient in the supine position looking directly upward and the pressure is registered on the scale. The normal pressure varies between 12 and 27.

Primary Glaucoma.—Primary glaucoma has a doubtful *etiology*, but several conditions are observed in eyes blinded by glaucoma which explain the increase of tension.

In order to interpret these conditions, the *drainage* of the aqueous chamber must be understood. The nutrient fluid secreted by the ciliary body to nourish the vitreous and the lens and replenish the aqueous chamber passes through the pupil into the anterior chamber and escapes from the eye by filtration through the pectinate ligament into Schlemm's canal and the venous system. The angle formed by the iris, the pectinate ligament, and the posterior wall of Schlemm's canal is known as the *filtration angle;* and it is evident that any condition which interferes with or obliterates this angle will prevent the drainage of the eye, whereby an accumulation of the aqueous will occur, and an increase of intra-ocular tension is the inevitable consequence.

In cases of primary glaucoma the filtration angle is either closed or considerably compressed, and the iris is either adherent to the cornea and pectinate ligament or pressed against them. In recent cases the ciliary processes and the ciliary muscle are drawn forward, but later they are atrophied and retracted.

The obliteration of the filtration angle is due primarily to pressure of the ciliary processes against the iris, and the explanation of increased tension is the retention of the intra-ocular fluid.

Acute Primary Inflammatory Glaucoma.—Acute primary inflammatory glaucoma is bilateral, one eye and then the other being affected, and occurs almost exclusively in individuals past forty years of age.

Etiology.—The eyes are frequently hypermetropic; sex does not predispose; heredity is often demonstrable; the Jewish race is predisposed; mental emotions are sometimes causal; sleeplessness, great and prolonged pain, starvation, respiration of foul air, excessive mental application, vomiting, and febrile diseases may lead to a depressed state of the body which favors glaucoma; myotics abort light cases and relieve severe ones, but do not prevent relapses; iridectomy restores normal tension and a sclerotomy is a palliative measure; an eye enucleated because of inflammatory glaucoma remains hard after enucleation; and the examination of an eye between the attacks of light glaucoma shows no pathological changes. The above facts represent what is positively known about acute inflammatory glaucoma.

Symptoms.—Acute primary glaucoma is sometimes preceded by premonitory symptoms, as dimness and rainbow vision; but the onset may be sudden and violent in the fulminating form. After some exciting cause, the eye becomes painful and a neuralgic pain radiates from the affected eye over the temple and side of the head. The lids are swollen and the conjunctiva is congested. The cornea becomes "steamy" from edema of its tissues, is not sensitive, and vision is much impaired. The pupil is usually dilated and does not react, and accommodation is diminished. The tension of the eyeball will be found more or less increased, and the anterior chamber becomes more shallow. Ophthalmoscopic examination, when possible, shows that the arteries are smaller and the veins engorged. Hemorrhages in the retina and choroid are occasionally observed. The blood-pressure is usually increased. A change of refraction of some dioptres is also observed in glaucoma. If *treatment* is not instituted, the sight is lost.

Subacute Primary Glaucoma.—Subacute primary glaucoma is characterized by a recurrence at intervals of imperfect and rainbow vision at the beginning; but later the increased tension is permanent, and its results, the glaucomatous cupping of the disk and contraction of the visual field due to insufficient nutrition of the retina, take place. Blindness is the result unless operative interference comes to the rescue, when some vision is still retained.

Absolute Glaucoma.—Absolute glaucoma implies that the eye is sightless, the tension is excessive, and the disk excavated. The cornea is transparent but insensitive, the iris reduced to a narrow band, and the anterior chamber is very shallow. Absolute glaucoma leads to ectasias of the sclera, particularly the equatorial form, opacity of the lens, and a haziness of the cornea. These manifestations are those of glaucomatous degeneration, which is often accompanied by pain. The final outcome may be atrophy of the bulb, or perforating abscess of the cornea, with its consequent iridocyclitis or panophthalmitis with phthisis bulbi.

Glaucoma Simplex.—Glaucoma simplex is a chronic primary affection, the only symptom of which is impairment of the sight. Increase of intra-ocular tension is not present at all times, and may be absent altogether. Chronic primary glaucoma, as it is also called, develops slowly and insidiously, and may require many years to destroy the sight. Like inflammatory glaucoma, it affects both eyes, one shortly after the other. Since pain and inflammation are not present, the patient may not be aware of the disease until the visual field becomes considerably contracted, the central vision remaining good for a long time. The eye may appear normal even when blind, or an inflammatory glaucoma may supervene with its results.

Hydrophthalmos or Buphthalmos.—Hydrophthalmos occurs either congenitally or shortly after birth, and since the sclera at this period of existence is elastic, an increase of intra-ocular tension may increase the size of the eyeball. In adults the lamina cribrosa is the only part of the sclera which yields to pressure, and this accounts for the presence of the glaucomatous excavation or cupping. Hydrophthalmos may come to a standstill, in which case the tension becomes normal and some sight is retained; or it may progress until the eye reaches frightful dimensions and sight is lost. Hydrophthalmos is usually a bilateral disease.

Treatment of Primary Glaucoma.—The treatment of primary glaucoma is palliative and operative. Physostigmine (better known as eserine) and pilocarpine, because of the myosis they produce, increase the permeability of the filtration angle and reduce the intra-ocular pressure. Cocaine dilates the pupil, but contracts the ciliary bloodvessels and diminishes the sensibility of the ciliary nerves, whereby the intra-ocular pressure is reduced. Cocaine in combination with physostigmine, the myotic action of which should dominate, is also useful. Atropine must be mentioned to be condemned in the treatment of primary glaucoma. It dilates the pupil, increases the intra-ocular pressure, and may not only precipitate an attack of glaucoma in a predisposed individual, but even destroy the sight in a short time when used during the disease. However valuable atropine is in the treatment of corneal and iritic disease, it is dangerous and disastrous in primary glaucoma.

Morphine, rest, warm nourishment, laxatives, and the local use of cold are valuable palliative measures in certain cases. The myotics are useful when the tension of the eye is notably increased, but of questionable utility in chronic glaucoma (simplex), which may not be accompanied by increase of tension.

An iridectomy, scleral trephine, cyclodialysis, anterior or posterior sclerotomy and iridosclerectomy are the *operations* suggesed for the relief or cure of primary glaucoma.

An *iridectomy* in order to be potent should be made in such a way that a large segment of iris is removed as near as possible to the ciliary attachment. The effect of a properly performed iridectomy upon the course of an acute inflammatory glaucoma is all the better for vision the sooner it is performed. An iridectomy is beset with some difficulty during this condition, owing to haziness of the cornea and shallowness of the anterior chamber, but its effect is sometimes curative. In chronic inflammatory glaucoma the result of the iridectomy will depend upon the duration of the cupping and atrophy of the optic nerve. Simple glaucoma is not so much benefited by an iridectomy, the most that can be expected being a retention of vision equal or nearly equal to that at the time of operation. Some cases actually do worse, and the eye is blinded after the iridectomy: these are the malignant ones.

An anterior sclerotomy, whereby an opening is made through the sclera into the anterior chamber, may be performed in place of the iridectomy, but is not so reliable.

An *enucleation* is indicated in absolute glaucoma when, on account of atrophy of the iris, an iridectomy cannot be performed or would be useless to relieve the pain.

Hydrophthalmos is subject to the same treatment as the other primary forms of glaucoma; but an iridectomy may be serious from the escape of the vitreous through the suspensory ligament, which is apt to tear when the anterior chamber is emptied.

Secondary Glaucoma.—Definition.—The increase of tension is due to some ocular disease which causes retention of the intra-ocular fluid.

Etiology.—An annular posterior synechia, by preventing communication between the posterior and anterior chambers, pushes the iris forward and the filtration angle is no longer permeable. Corneal wounds or ulcers when associated with anterior synechia interfere with the filtration angle according to the amount of iris adherent to the cornea. When the cornea is perforated, establishing a fistula, the intra-ocular pressure is not increased so long as the aqueous can leak out of the corneal opening; but closure of a fistula of long duration may cause increased tension. A serous cyclitis, from the altered character of the secretion, may cause an accumulation of morbid fluid which is too thick to filter through the pectinate ligament in the anterior chamber, the result of which is a deepening of the anterior chamber and widening of the filtration angle. After cataract operations secondary glaucoma may develop at a variable period of time. The filtration angle is always found to be abnormal. A dislocated lens may produce increased tension; and a discission or wound of the lens, because of the swelling of the lens, may have a similar effect. Intra-ocular tumors at a certain stage produce intra-ocular pressure, and the filtration angle will be found disturbed in these cases. Exudations in consequence of iridocyclitis or choroiditis sometimes lead to increase of tension, and hemorrhages into the vitreous may have a like result. Ectasia of the sclera is at times responsible for increase of tension.

Treatment.—The treatment of secondary glaucoma depends, of course, upon the cause. An annular posterior synechia requires an iridectomy to establish communication between the posterior and anterior chambers. Corneal staphyloma may be treated by excising the bulging part. Ectasias of the sclera producing glaucoma are best treated by an iridectomy if it be possible; otherwise by enucleation when the pain is considerable. The tension due to a serous cyclitis is relieved by a paracentesis repeated several times if necessary. A dislocated or swollen lens should be extracted when causing glaucoma, if it be possible. Tumors require enucleation of the eye; and hemorrhagic glaucoma is usually not amenable to other treatment. All sightless and painful eyes indicate enucleation.

THE LENS.

ANATOMY.

The **crystalline lens** is a transparent, lenticular body, biconvex in shape, the posterior curvature being the greater.

It is enclosed in an elastic envelope, or **capsule**, the anterior part being more than twice as thick as the posterior, and hence more subject to pathological conditions. The anterior part of this envelope is known as the *anterior capsule*, the posterior part as the *posterior capsule*, although the envelope is the same in structure. It is to the elasticity of the capsule that the lens owes the property of changing its curvature, whereby the rays of light may be brought to a focus on the retina in adaptation of the eye for sight at different distances.

Equator and Axis.—The *equator* of the lens represents the margin at which the lenticular surfaces meet. The antero-posterior *axis* of the lens extends from the so-called anterior to the posterior pole, which is at the junction of the anterior and middle thirds of the optical axis of the eye.

The lens is held in place by the **suspensory ligament**, or zonula, whose lenticular attachments are: in front, to the anterior capsule near the equator; behind, to the corresponding margin of the posterior capsule.

The annular space, triangular on section, bounded by these anterior and posterior lamellæ of the zonula and the equatorial margins of the lens, is the *canal of Petit* of the older anatomists.

Accommodation.—The lens and zonula separate the aqueous and vitreous humors. When the zonula exerts traction on the capsule, the convexity of the lens is diminished; when it is relaxed the convexity is increased. These changes of the lens constitute accommodation. The lens up to adult life has throughout the same consistence. The central part, known as the nucleus, then becomes denser than the external, or cortical, part. With increasing age, the lens substance becomes less pliable, and on this account the elasticity is less and the power of accommodation diminishes.

The **substance of the lens** consists of lens fibers, each of which is composed of two layers of epithelium. These ribbon-like fibers are so arranged that a part of each is on the anterior as well as the posterior surface. They are kept in apposition by a cement substance, and the lines of apposition constitute the lens stars, which can be better observed in young subjects than in adults.

The *lens stars* on the anterior surface of the lens resemble an inverted Y (⅄); those on the posterior surface the erect letter Y. The lens fibers forming the inner and middle portions of the lens have serrated edges; the peripheral lens fibers are smooth and have nuclei. The *minute spaces* between the fibers contain the semifluid cement substance, and these channels afford passageways for the transmission of the nutritive fluid to the fibers of the lens. The ciliary body secretes the fluid for nutrition of the lens, and this enters near the equator.

The **substance of the suspensory ligament,** the zonule of Zinn, or the zonula ciliaris, is composed of transparent homogeneous connective-tissue fibers, which arise largely from between the folds of the ciliary processes and also from the ora serrata and the smooth surface of the ciliary body. The fibers which arise posteriorly are inserted on the posterior surface of the lens; the others cross in such a way that the more anterior fibers are inserted on the posterior surface and the posterior fibers go to the anterior surface of the lens.

In **accommodation**, the elastic lens, the suspensory ligament, and the ciliary body are concerned. When the eye is accommodating for the near point, the lens, because of its elasticity, assumes its greatest convexity, the suspensory ligament being slack from the position of the ciliary body, which during this act moves forward and inward. When the ciliary muscle is at rest, the ciliary body makes traction upon the fibers of the suspensory ligament, and in consequence of this the convexity of the lens is diminished and the eye is adapted for distant vision.

MALFORMATIONS OF THE LENS.

Congenital Absence. — Congenital absence of the lens (aphakia) is rarely seen, and then mostly in microphthalmic eyes.

Lenticonus.—Lenticonus is a rare anomaly in which the anterior surface (less often the posterior surface) presents a conical projection. After the use of a mydriatic, an ophthalmoscopic examination reveals a central red, circular area surrounded by a narrow, ring-like shadow, external to which is an illuminated zone.

Coloboma.—Coloboma of the lens sometimes is associated with a congenital coloboma of the iris. The lens is more or less defective at the site of the coloboma iridis.

Ectopia Lentis Congenita (*subluxation of the lens*) is often hereditary. It is due to a difference of width of the suspensory ligament, whereby the lens is not held in proper position. A congenitally dislocated lens is sometimes cataractous at birth or may become so later. A partial dislocation very often becomes a complete one, when the iris will be found tremulous. The *treatment* depends upon the patient's vision, operation (removal of the lens) being only undertaken when other measures (those for correcting refraction) are unsuccessful. When the dislocated lens is producing irritation and cannot be removed, enucleation is indicated.

Congenital Cataracts.—Congenital cataracts involve either the entire lens or a portion of it.

Complete cataracts may be fluid, gelatinous, or hard in consistency. Discission is the treatment, and this may be done when the patient has lived three or four months. In hard and shrunken cataracts, discission is not successful, since the membrane remaining after operation always remains opaque.

The *partial cataracts* which may be congenital are *anterior polar*, *posterior polar*, *perinuclear*, and *dotted*.

Dotted cataracts are apparent as small spots or streaks in the cornea, and are often hereditary. The dots occur mostly in the periphery of the lens, and do not occasion any disturbance of vision: therefore they require no treatment.

A *posterior polar cataract* is an opacity of the posterior capsule at the central part, due to the presence of the hyaloid artery, which extends across the vitreous chamber in fetal life. No *treatment* is called for.

The other forms of partial cataract will be considered under *Cataracts*.

INJURIES OF THE LENS.

Dislocation.—A traumatic dislocation of the lens usually occurs as a result of contusion of the bulb. In some extensive ruptures of the bulb the lens may be expelled with other of the contents and the eye is usually lost.

Treatment.—When suitable glasses assist the vision, these should be prescribed. When symptoms of secondary glaucoma or iridocyclitis occur, the lens should, if possible, be extracted. This is easy when the lens is in the anterior chamber, difficult or impossible in a partial dislocation, and quite impossible when the lens floats in the vitreous. When removal of the lens is not practicable, an iridectomy may relieve the tension or the inflammation. When a sightless eye is painful and inflamed because of a dislocated lens, an enucleation relieves the patient and prevents a possible sympathetic inflammation.

Traumatic Cataracts.—Traumatic cataracts are due to an infinite variety of injuries. Simple concussion, lightning-stroke, penetrating wounds of the bulb, associated or not with concussion, or the entrance of a foreign body into the eye may produce cataracts.

Prognosis of Injuries of the Lens.—The prognosis of injuries of the lens depends upon the presence of a foreign body in the eye, infection, iridocyclitis, secondary glaucoma, and the situation and extent of the wound.

Treatment of Injuries.—A traumatic cataract may be treated by discission or extraction, according to the patient's age. When the lens capsule is torn, the lens substance should be left alone and allowed to absorb, unless its presence indicates danger by irritation or increase of tension, when extraction is indicated.

When traumatic cataracts are not complicated by wounds of the ciliary region or the sclera, detachment of the retina, rupture of the choroid, or iridocyclitis, the removal of the lens offers good hope for vision. When foreign bodies enter the eye and remain imbedded in the lens, the removal of the lens should be undertaken as soon as it is ripe. An involvement of the iris to any extent is prejudicial to success in the management of a traumatic cataract. A prolapsed iris should be cut off. The integrity of the capsule need not necessarily be impaired in traumatic cataracts, yet in most cases laceration of the capsule, exposing the lens substance to the action of the aqueous, occurs.

Cataracts.—**Definition.**– An opacity of the lens is known as a cataract.

The following kinds of cataract, classified by the location of the opacity, are seen: anterior polar; posterior polar; zonular; anterior and posterior cortical; and nuclear.

Besides these, senile, congenital, secondary, Morgagnian, and traumatic cataracts occur.

All cataracts are partial or complete, or stationary or progressive.

Anterior Polar Cataract.—The anterior polar cataract is located between the anterior capsule and the lens at its anterior pole, and appears like a small white spot. It may be congenital or acquired.

When *congenital*, it is usually bilateral and due to faulty development of the lens.

The cause of the *acquired* form is a central ulceration of the cornea, with perforation and loss of the aqueous humor, whereby the anterior capsule comes into contact with the cornea and produces opaque tissue under the capsule.

When the cataract is so large that it causes a noticeable projection of the anterior pole of the lens, it is known as a *pyramidal cataract.*

These cataracts can be seen by oblique illumination. On examination with the ophthalmoscope, it is noticed that there is no parallactic displacement, which shows that the cataract is near the plane of the pupil.

Treatment.—Treatment is usually not required. When of very large size an iridectomy is indicated.

Zonular, Perinuclear, or Lamellar Cataract.—A zonular, perinuclear or lamellar cataract is due to opacification of the layers of the lens between the cortex and nucleus. Only a few lamellæ may be involved, in which case spiculæ, or spokes, are seen; or there may be a diffuse opacification which appears like a ring. It may be congenital or may form in early infancy. It is usually bilateral and stationary.

These cataracts are often observed in individuals who show evidences of rhachitis.

Treatment.—Treatment is usually not required. When vision is considerably interfered with, an iridectomy is indicated, if the periphery of the lens be normally transparent; otherwise an extraction or discission of the lens, according to the age of the patient, is necessary.

Anterior or Posterior Cortical Cataract.—When opacities occur in the anterior or posterior layers of the cortex the condition is known as anterior or posterior cortical cataract.

The *posterior* form is more common than the *anterior*, but both may be present at one time.

These cataracts are usually due to disturbance of nutrition, as a result of disease of the vitreous humor, choroid, or retina. They remain stationary for years, but finally a total opacity occurs. Because of the damaged condition of other parts of the eye, vision is bad, even after operation, which should not be too readily undertaken.

Senile Cataract.—The senile cataract is the most common of all forms, and usually begins to develop after the fiftieth year. It commences as a partial opacity, which increases in size until the entire lens is involved. Both eyes are frequently affected, one a variable time after the other.

A senile cataract is *due* simply to the degenerative changes incident to old age, and not to morbid conditions, although certain diseases like diabetes may produce cataracts.

The *development* of a cataract from its incipiency to maturity or complete opacification requires a variable period of time—from a day to twenty years.

The senile cataract, being progressive, is divided into four stages, each of which has its characteristics and significance.

The *first stage*, or that of incipiency, presents upon examination spiculæ, or other form of opacity, which involve only a portion of the lens. The anterior chamber is of normal depth.

The *second stage*, or that of intumescence or swelling, is characterized by an increase in volume of the lens, whereby the anterior chamber becomes more shallow than normal. When a candle is held to one or the other side of the eye, the iris throws a shadow through a part of the lens, indicating that this part of the lens is transparent. The lens star is plainly visible, the lens has a bluish-white color, and its surface appears smooth, glossy, and silky.

The *third stage*, or that of maturity, is characterized by the normal depth of the anterior chamber, due to the loss of water which in the second stage caused the intumescence. The lens is completely opaque, in consequence of which the iris no longer throws a shadow. The lens is of a dull gray or brown color, and the lens star can still be discerned. This is the stage for operative interference, since the lens substance can be removed *en masse* from the capsule.

The *fourth stage*, or that of hypermaturity, presents an abnormally deep anterior chamber, the iris throws no shadow, and the lens substance has liquefied.

Senile cataracts are hard until they become hypermature or overripe, when they are soft.

When the nuclear portion of the lens remains hard and the rest is liquid, a so-called *Morgagnian cataract* results.

An overripe cataract is subject to change from the deposition of cholesterin or calcareous material, or the anterior capsule may become thickened, forming a so-called *capsulo-lenticular cataract.*

Progressive Cataracts.—Progressive cataracts are often due to the poor nutrition incident to general diseases, as diabetes. The cataracts are bilateral and usually develop with great rapidity.

Secondary Cataracts.—Secondary cataracts are those which are the result of some ocular affection. The cataract receives its nutrition largely from the ciliary body, and hence any disease affecting its secretory function will interfere with the nutrition of the lens. Glaucoma, retinitis pigmentosa, myopia of high degree, iridochoroiditis, detachment of the retina, and iridocyclitis may lead to opacification of the lens.

A *secondary capsular cataract*, or membranous cataract, is the result of traumatism or operation when parts of the lens substance and capsule remain in the eye and begin to proliferate. The sight may be much reduced in spite of endeavors to keep the pupil clear.

Cataract Nigra.—Cataract nigra, or black cataract, gives the pupil the normal dark appearance, but ophthalmoscopic examination shows that the lens is not transparent. It may be due to infiltration of blood, or to a condition of the lens in which the cortical is changed into nuclear tissue, which undergoes degenerative changes.

THE VITREOUS HUMOR.

ANATOMY.

The vitreous humor, or hyaloid body, is a gelatinous, transparent substance which fills the space bounded by the lens in front, and the retina at the sides and behind. It represents four-fifths of the space of the eyeball, and is inclosed in a thin, transparent hyaloid membrane, which is, however, wanting at the lenticular fossa in which the posterior capsule of the lens rests.

The **hyaloid membrane** is attached to the retina (to the anterior part much more firmly than to the posterior), the ciliary body, the zonula, and most firmly to the optic disk, in consequence of the entrance of the hyaloid artery during fetal life at this point.

The **canal** which extends from the papilla to the posterior pole of the lens is known as Cloquet's (or Stilling's) canal. In fetal life the hyaloid artery courses through this canal, which after birth is probably a lymph canal, the artery having disappeared. The hyaloid artery should present, if anything, only a trace after birth.

Uses.—The vitreous prevents detachment of the retina and dislocation of the lens, and maintains the shape of the eye.

The vitreous has no bloodvessels, and receives its nourishment from the uvea, more particularly from the ciliary body.

CONGENITAL ANOMALIES.

In fetal life the vitreous contains many cells, and is therefore quite opaque. At times some of these cells remain, producing **slight opacities,** which give, however, very little trouble.

A **persistence of the hyaloid artery** sometimes occurs, and is recognized as a gray cord extending from the papilla a variable distance into the vitreous, sometimes even to the posterior capsule of the lens. The canal of Cloquet is visible when the walls are for some reason opaque.

Posterior polar and cortical **cataracts** occasionally complicate these conditions.

INJURIES OF THE VITREOUS.

Foreign Bodies.—Foreign bodies projected into the vitreous or a lens dislocated from injury usually cause considerable inflammation: at times an iridocyclitis, purulent hyalitis, or panophthalmitis. Foreign bodies should be removed as soon as possible. A magnet may be employed to remove pieces of iron with some chance of success. Failing to remove the foreign body, an enucleation must be done to prevent sympathetic inflammation.

Hemorrhages.—Hemorrhages into the vitreous produced by injury cause more or less opacity. If the hemorrhage be not too great, absorption may take place. The iodides and diaphoretics are possibly of some service.

DISEASES OF THE VITREOUS.

Opacities.—Opacities of the vitreous are the result of exudates due to inflammations of the uvea or retina or spontaneous hemorrhages. The patient notices floating particles (muscæ volitantes), visual acuity being diminished in proportion to the number of the opacities.

Prognosis.—The prognosis depends upon the duration of the exudates.

Treatment.—Recent exudates are often amenable to treatment; old ones are not. The iodides and eliminants generally are useful in some cases.

Sometimes the **exudate** actually fills the vitreous chamber. A *plastic exudate* may become organized and result in atrophy of the bulb. A *suppurative exudate* usually terminates in rupture of the sclera, with loss of some of the contents of the eye—phthisis bulbi.

A *purulent hyalitis* is a purulent infiltration of the vitreous, but cannot be recognized by a yellow reflex seen through the pupil except in mild cases. The condition terminates in panophthalmitis and phthisis bulbi. *Treatment* is useless.

Synchisis of the vitreous is a liquefaction of more or less of the vitreous. This condition *complicates* diseases of the retina and choroid. It may lead to atrophy of the bulb from a diminution of the quantity of the vitreous; detachment of the retina; and softening of the zonula, which may later permit spontaneous luxation of the lens.

Parasites.—Cysticercus cellulosæ and filaria are very rarely found in the vitreous. They should be removed, since shrinking and loss of the eye are the inevitable result. If removal be impossible, enucleation is indicated.

THE RETINA.

ANATOMY.

The retina is the inner of the three coats of the eye. It lies between the vitreous and the choroid. Because it is more firmly adherent to the vitreous than to the choroid, detachment of the retina from the choroid may occur in consequence of a loss of vitreous. The retina extends from the entrance of the optic nerve to the posterior end of the ciliary body, ending, with the exception of the tapetum, in a wavy line, the *ora serrata*.

The part of the retina between these points is known as the *pars optica retinæ*, and is of the greatest importance. The *tapetum*, or pigment-layer of the retina, forms a single layer of epithelial cells on the inner surface of the ciliary body, and this part is known as the *pars ciliaris retinæ*. From the ciliary body this layer passes to the iris, lining its entire posterior surface. This part is known as the *pars iridica retinæ*.

The retina is quite transparent: it is thickest near the disk, where it measures $\frac{1}{75}$ of an inch, and thinnest near the ora serrata, where it is $\frac{1}{200}$ of an inch in thickness.

The retina is composed of ten layers from within outward, as follows: (1) the internal limiting membrane; (2) layer of optic nerve fibers; (3) layer of ganglion cells; (4) internal plexiform, or molecular, layer; (5) internal nuclear, or layer of bipolar cells; (6) external plexiform, or molecular, layer; (7) external granular, or layer of bodies of visual cells; (8) external lining membrane; (9) layer of rods and cones; and (10) the pigment layer. The internal and external limiting membranes and the radiating fibers of Müller constitute the support for the nervous part of the retina.

The **internal limiting membrane** is a kind of connective tissue formed by the cone-shaped extremities of the supporing fibers of Müller.

The **external limiting membrane** represents the external extremities of Müller's fibers.

The **macula lutea** is an oval spot situated one-eighth of an inch to the outer side of the papilla. At its central point is a slight depression, known as the *fovea centralis,* which is the most sensitive part of the retina. The macula represents the centre of direct vision, and the papilla, which is a little to the nasal side, has fibers which are insensible to light, and hence in the field of vision there is a corresponding blind area, known as the "*blind spot* of Mariotte" (Plate IV).

The **arteria centralis retinæ** supplies the inner layers of the retina with blood. This vessel divides into four branches, called the superior and inferior nasal and the superior and inferior temporal. These arteries are accompanied by a corresponding vein, and do not anastomose, thereby causing much trouble when for any reason an artery becomes plugged.

The *fovea centralis* and the external layers of the retina receive their nutrition from the capillary layer of the choroid, which provides nutritive lymph.

In **normal eyes** a light streak is seen on the retinal vessels. This is supposed to be due to refraction of rays of light in passing through the bloodvessel, the posterior wall of which and underlying tissue reflecting the rays back to the observer.

ANOMALY AND NEOPLASM OF THE RETINA.

Opaque Nerve Fibers.—Opaque nerve fibers are not, as is generally described, a congenital anomaly, since fibers in any case at birth have no medullary sheaths for some distance behind the lamina cribrosa. In the case of opaque nerve fibers, the medullary sheaths continue their course beyond the lamina cribrosa into the bulb, and in consequence one sees on ophthalmoscopic examination white spots starting usually at the margin of the disk and ending in a brush-like extremity. The interference with vision depends upon the size and position of the patches. The blind spot is increased in size.

There is no *treatment.*

Glioma.—The only neoplasm of the retina is *glioma;* and this occurs only in children. One or both eyes may be involved, but usually the trouble is unilateral. This neoplasm is similar to a sarcoma of the choroid in its development, and accordingly four stages are described. In the ***first stage***, the pupil presents a peculiar reflex and the eye is sightless, but there is no external sign of inflammation. In the ***second stage***, the eye becomes glaucomatous, with the attending pain and irritation. In the *third stage*, the growth perforates the bulb. In the *fourth*, metastatic invasions, more particularly in the liver, occur.

An early enucleation offers the only chance for life. Exenteration of the orbit is sometimes indicated, but is too often useless.

INJURIES OF THE RETINA.

Traumatic Detachment of the Retina (Fig. 23).—Traumatic detachment of the retina may be *produced* by concussion, with or without opening of the bulb, and by foreign bodies. Hemorrhages from the choroidal vessels may push the retina away from the choroid, since the retina is firmly attached only to the external tissues at the disk and ora serrata. The pressure of the vitreous under normal circumstances keeps the retina in contact with the choroid, and any considerable loss of vitreous diminishing the pressure permits a retinal detachment.

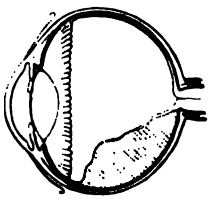

FIG. 23.—Section of eye with partial detachment of retina. (Nettleship.)

Rupture of the Retina.—Rupture of the retina sometimes occurs as a result of a diminished amount of vitreous, which in shrinking tears the retina, to which it is attached. The vitreous may then pass through the rupture, and thus separate the retina from the choroid. Rupture of the retina seldom occurs from concussion.

Diagnosis.—The diagnosis of *detachment* is only difficult when the vessels cannot be recognized upon the veil-like retina. A difference of refraction of different parts of the retina is demonstrable.

Rupture of the retina can be diagnosed by the interrupted course of the vessels.

Prognosis.—The prognosis of traumatic detachments is not hopeless. In eyes previously sound, the retina may become reattached and the vision may not suffer to any great extent.

Treatment.—As soon after the injury as possible the eyes should be bandaged, and the patient should remain on his back for some weeks.

Concussion.—Concussion of the retina is due to blows upon the bulb. The retina has a milky appearance for a day or two, after which, if anything, only a few hemorrhagic spots are left.

Foreign Bodies.—Foreign bodies sometimes remain fastened in the retina. They are first covered by blood, and finally become imbedded in a connective-tissue capsule.

They should be removed.

Traumatism may cause *rupture of the retinal vessels*, and some retinal hemorrhages have their origin in this way.

Pathologic Detachment of the Retina.—Pathologic detachment of the retina may be *caused* by spontaneous hemorrhages from the retina, retinitis (more particularly albuminuric), myopia with posterior staphyloma, intra-ocular tumors (especially sarcoma of the choroid), cysticercus subretinalis, orbital abscesses and tumors, or chronic inflammations of the choroid.

The *prognosis* is in general bad, but is modified by the condition of the choroid and vitreous.

Some favorable cases do well on diaphoretics and absolute rest in the recumbent position. When detachment becomes complete, enucleation is necessary to relieve pain and prevent sympathetic inflammation.

DISTURBANCES OF BLOOD SUPPLY.

Hyperemia.—Hyperemia of the retina may be active or passive.

Active hyperemia is observed in the initial stages of inflammation of the optic nerve or retina. Meningitis, syphilis, tumors of the orbit or optic nerve, sunstroke, and a high degree of hypermetropia may produce active hyperemia.

The veins are seen to be enlarged and tortuous, and the reflexes are interrupted, since successive parts of the vein do not lie in the same plane; the arteries remain unchanged.

Passive hyperemia accompanies glaucoma, papillitis, orbital tumors, and general anemia.

Anemia.—Anemia of the retina may be due to loss of blood, general anemia, an embolus of the retinal vessels, and retrobulbar neuritis. Quinine or bromine amaurosis is attended with anemia of the papilla and retina.

Hemorrhages.—Hemorrhages of the retina may take place in either the external or the internal layers. When in the *external layers*, the hemorrhages are round or irregular in form; when in the *internal layer* of nerve fibers, they appear in streaks, since the blood follows the course of the fibers.

Hemorrhages may be *due* to traumatism, atheromatous degeneration of the bloodvessels, extreme anemia, active and passive hyperemia of the retina, embolus of the central artery, thrombosis of the central vein or of its branches, or local disease of the retinal and the adjoining choroidal vessels.

Retinal hemorrhages are absorbed in a few months, and the existence of a scotoma depends upon the extent of damage done to the retinal structure.

A *total embolus* of the central artery produces marked anemia of the retina, the arteries being reduced so much in size that they appear like thin threads. About the papilla they may be easily seen, but at a variable distance from it they may hardly be recognized. The veins are somewhat diminished in size, and the papilla is quite white and its margin is indistinct. The nutrition of the retina being interfered with, it becomes opaque to the extent of surface containing the external plexiform layer. Because this is wanting in the fovea centralis the normal red color of the fundus is here preserved.

Sudden blindness is the history given in cases of total embolism.

In a few recent cases the embolus may advance into a small branch of the central artery and a part of the retina regain its function. Generally the sight is permanently lost, since atrophy of the retina and papilla soon follows the other condition.

When the embolus is infected, a suppurative retinitis and finally a panophthalmitis result.

An embolus lodging in a branch of the central artery produces a disturbance of vision corresponding with the area involved. The retinal changes occur only in that part supplied by the vessel affected.

Thrombosis.—Thrombosis of the central vein when complete causes great disturbance of vision, which soon becomes total loss of sight. The veins are tortuous and enlarged, the arteries hardly visible, the papilla is generally congested, and retinal hemorrhages are seen in all parts of the fundus. Thrombosis of the smaller branches may occur, and this produces the changes already mentioned limited to the affected part. An orbital cellulitis (more particularly when caused by facial erysipelas), valvular disease of the heart, and atheromatous degeneration of the bloodvessels may cause thrombosis.

INFLAMMATIONS OF THE RETINA.

Retinitis Externa or Interna.—According to the seat of the inflammation, whether it be in the outer or the inner layers of the retina, we distinguish retinitis externa or interna.

A *retinitis externa* is *caused* by general diseases, such as syphilis, which so frequently involves the choroid; and local diseases of the eye, such as myopia, which produces choroidal changes.

A *retinitis interna* is *caused* by diseases which produce degenerative changes of the vessels or a change in the composition of the blood. A *hemorrhagic retinitis* is due to the former cause; an *albuminuric*, *leukemic*, or *diabetic retinitis* to the latter.

A *retinitis externa produces changes* of the pigment layer, appearing as whitish atrophic spots, which, if the choroid be also atrophied, permit a view of the sclera. Accumulation of pigment frequently occurs at the borders of these atrophic spots. A retinitis externa is nearly always *associated with* a choroiditis, which is generally the primary disease.

A *retinitis interna is recognized* by a hyperemia and cloudiness. The papilla is indistinct, sometimes swollen; extravasations and whitish masses occur in the supporting fibers and in the optic nerve fibers; and the walls of the retinal vessels may suffer a change.

Retinitis Syphilitica.—Retinitis syphilitica is the commonest form of retinitis, and is usually associated with disease of the choroid and iris. The disease may be circumscribed or diffuse.

When *circumscribed*, the exudate is seen near a large retinal vessel or in the macular region. The absorption of the exudate produces scars, which may by contraction cause a detachment of the retina.

In the *diffuse* form the retina is opaque, more particularly near the larger vessels. Lymph corpuscles infiltrate the inner retinal layers and the supporting fibers become thickened. Bluish-white masses, often larger in size than the papilla, occur, especially in the macular region, and the retina is much thickened in the affected parts. The inflammatory products may become absorbed, leaving spots under which the choroidal vessels are seen and around which is a deposition of pigment.

Atrophy of the retina sometimes occurs, and this is diagnosed by the small size of the vessels and the change of the color and indistinctness of the papilla.

The *choroid* and the *vitreous* are very prone to become involved in a syphilitic retinitis. Small dust-like particles seen in the vitreous are due to implication of the vitreous.

Treatment.—Antisyphilitic treatment should be instituted, although much cannot be promised.

Retinitis Albuminurica.—Retinitis albuminurica is nearly always a bilateral disease characterized by haziness of the retina, indistinct margin of the papilla, hemorrhages, and white spots occurring about either the papilla or the macula. The white spots frequently present a stellate arrangement about the macula, and thus make this form of retinal disease characteristic.

The disease is *caused* by changes in the vessels induced by the condition of the blood incident to renal disease.

Prognosis.—The prognosis of retinitis albuminurica is grave, except when caused by scarlatinal nephritis or parturition.

Retinal detachment occasionally occurs when the disease is severe. The detached part is opaque and fixed, and is globular in shape.

Retinitis Leukemica.—Retinitis leukemica is characterized by pallor of the fundus, retinal hemorrhages, and white patches due to the exudation of white and red blood corpuscles. Sometimes the veins appear red and the arteries yellow. White patches with red borders occurring near the equator and in the macular region are of diagnostic value in connection with pallor.

Retinitis Diabetica.—Retinitis diabetica can be *differentiated* from retinitis albuminurica by the presence of groups of small semicircular or serrated bright spots occurring between the superior and the inferior temporal branches of the central artery, by the absence of the stellate arrangement of spots about the macula, by limited opacity of the retina, and by a normal papilla.

Retinitis Hemorrhagica.—Retinitis hemorrhagica is the result of changes occurring in the retinal vessels. Heart disease, gout, atheromatous or calcareous degeneration of vessels in general, pernicious anemia, syphilis, and sepsis may lead to retinal hemorrhages. These hemorrhages often terminate in atrophy of the retina and papilla.

Sclerosis of the Retina.—Sclerosis of the retina, so-called *retinitis pigmentosa*, is a bilateral disease, sometimes congenital, frequently hereditary, characterized by a slow and progressive fibrous tissue degeneration with or without a deposition of pigment. The papilla is rather yellow, its margin is very distinct, the retinal vessels are much reduced in size, and the retina itself is no longer transparent, but appears like a veil over the choroidal vessels. *Pigmentation* at first occurs in the periphery of the fundus, and progressively invades the more central parts. The pigment spots appear not unlike bone corpuscles, occurring more particularly along the sides of the vessels.

The patient has frequently malformations not only of the eyes, but also of other parts of the body: since a third of the cases are the offspring of parents who are blood relations, *consanguinity* would seem to be an important *cause* of the disease.

Night-blindness (hemeralopia) is the most important symptom. A concentric contraction of the visual field, but with fairly normal central vision, occurs in a typical case. When the vision is poor from the beginning, nystagmus, which renders examination difficult, is often present. A posterior cortical cataract is a frequent complication; it may appear as a dot, a disk, or a star.

Diagnosis.—Retinitis pigmentosa might be confounded with a *choroiditis* or a *retinochoroiditis syphilitica.*

The pigmentary spots in choroiditis are traversed by the retinal vessels, which in retinitis pigmentosa are hidden because the deposit occurs in the inner layer of the retina. Choroiditis is associated with atrophic changes which do not occur in retinitis pigmentosa. In retinochoroiditis, the outline of the choroidal vessels, apparent through the atrophied retina, and the pigment spots are to be seen as in retinitis pigmentosa; yet opacities in the vitreous and choroidal changes in the periphery and in the macular region are differentiating points. In some cases the history is the only means of diagnosis.

Treatment.—Treatment is of no avail. It is impossible to check or improve the condition, and the only comfort that can be given is the fact that the disease advances slowly. The patient should, however, be instructed to protect and rest the eyes.

THE OPTIC NERVE.

ANATOMY.

The **nerve fibers** which form the retina collect at a place a short distance to the nasal side of the posterior pole of the bulb, constituting the papilla, or optic disk, which can usually be seen with the ophthalmoscope, and is therefore an invaluable landmark in making ophthalmoscopic examinations.

The **disk** is a round or oval whitish spot that has a diameter of about 1.5 millimeters ($\frac{1}{18}$ of an inch). It appears nearly sixteen times larger upon direct, and four times larger upon indirect, ophthalmoscopic examination. The papilla is often surrounded by a whitish circle, known as the *scleral ring*, which represents the exposed edge of the sclera. A blackish circle exterior to this, known as the *choroidal ring*, is due to the view of the pigment which is sometimes present.

The nerve fibers coming from the constricted head of the optic nerve diverge in all directions in the formation of the retina, causing an **excavation** of the disk. This is but exceptionally in the centre of the disk, and can be recognized by its exceeding whiteness. This excavation is physiological and is not to be confounded with pathological excavations, which may be either glaucomatous or atrophic. The **glaucomatous excavation,** or cupping, differs from the **atrophic** in that it is deeper and the lamina cribrosa is pushed back. In **physiological excavations** a part of the disk is involved, whereas pathological excavations concern the entire disk.

The **intra-ocular portion** of the optic nerve is the papilla or disk, and extends back to the lamina cribrosa, which consists of the inner layer of the sclera and the modified choroid. The **lamina cribrosa** presents many perforations for the passage of the nerve fibers, which up to this point only have sheaths. The weakest and most yielding portion of the tunics is the lamina cribrosa, and accordingly an increase of intra-ocular tension is first manifest by bulging at this place. Moreover, the sclera surrounding the intra-ocular portion of the nerve is unyielding, so that swelling of the optic nerve causes a constriction interfering with the nutrition of the retina, of which the internal layers are suplied by the arteria centralis retinæ.

The **central artery** and **vein** pass through the middle of the intra-ocular portion of the optic nerve, and pathological changes of this part cause changes of circulation and nutrition which may impair the sight.

The **orbital portion** of the optic nerve extends from the lamina cribrosa to the optic foramen. Its length exceeds the distance from the optic foramen to the eyeball, so that the movements of the eye are not interfered with.

The *sheaths* of this part of the optic nerve are three, corresponding with the pia, arachnoid, and dura mater. The space between the arachnoid and dura is the subdural space; that between the arachnoid and pia the subarachnoid space. These spaces communicate posteriorly with similar spaces between the meninges of the brain, and end anteriorly in blind extremities in the sclera.

The **intracranial portion** of the optic nerve extends from the optic foramen to the chiasma, and is about one-third of an inch in length. It has but the pial sheath, the others covering the nerve at its exit from the optic foramen.

The optic nerves unite in the **chiasma** in such a way that about three-fifths of the nerve fibers of each nerve cross to the other side. Accordingly, only two-fifths remain on the same side. The decussating fibers supply the nasal half of the eye, the others the temporal half.

The optic nerve-fibers from the chiasma to their origin in the brain form the **optic tracts**. Some nerve fibers have been traced to the cortical substance of the occipital lobe, others (Meynert's communicating fibers) to the floor of the Sylvian aqueduct to the nucleus of the origin of the motor oculi. From the intimate relation of the motor oculi to the optic nerve fibers, it is easy to understand why light should cause a reflex contraction of the pupil, and why both pupils contract consensually, some optic nerve fibers of a single eye passing to the nuclear centres of both of the third nerves.

MALFORMATIONS AND NEOPLASMS.

Congenital malformations of the optic nerve:

A *coloboma* of the sheath of the optic nerve may be partial or complete. The cleft upon which the existence of the coloboma depends is in the under surface of the nerve, and accordingly a partial coloboma occurs at this place.

Congenital atrophy of the optic nerve produces the same picture as postnatal atrophy.

Congenital glaucoma always produces deep cupping of the disk, which can be easily demonstrated with the ophthalmoscope.

The *papilla* is sometimes apparently *reversed,* the temporal pole being turned toward the nose.

Endotheliomas, various kinds of **sarcoma, gliomas, fibromas, neurofibromas, tubercle,** and **gummata** have occurred in the optic nerve, and may not produce characteristic appearances.

INJURIES OF THE OPTIC NERVE.

The optic nerve may be injured by *foreign bodies,* as bullets; or *cuts* which involve the orbital structures.

Hyaline concretions are rarely found in the tissues of the papilla.

Injuries of the skull, either concussion or fracture, may damage the optic nerve, which after a time becomes atrophic.

Unfortunately little or nothing can be done in cases of optic-nerve injury.

INFLAMMATIONS OF THE OPTIC NERVE.

Inflammations of the optic nerve are classified as *intraocular,* when the papilla shows evidences of disease; and as *retrobulbar,* when the disease is confined to the orbital portion of the nerve.

Papillitis.—Definition.—A *papillitis,* or inflammation of the intra-ocular portion of the optic nerve, is a chronic disease characterized subjectively by disturbance of vision and objectively by intra-ocular changes.

Ophthalmoscopic Picture.—The disk is changed in color, its margin is indistinct, and it appears enlarged. The arteries are smaller, and the veins larger and tortuous because of their compression by the swollen nerve head between the unyielding sclera. The papilla is actually swollen, so that it extends farther forward than usual.

When the papilla does not show signs of great inflammation and the circumpapillary portion of the retina is involved, the condition is called a *papilloretinitis*. In this disease the ophthalmoscopic picture is that of a mild papillitis with exudates in the circumpapillary part of the retina; the vessels may be but slightly changed in appearance.

Etiology.—A papillitis may be due to intracranial, orbital, systemic, or localized diseases; and to injuries or primary affection of the nerve.

The *intracranial causes* of papillitis are basilar meningitis, either traumatic, tubercular, syphilitic, pyemic (especially from disease of the middle ear), or epidemic cerebrospinal meningitis; cerebral (extrameningeal and intrameningeal) abscesses; tumors; aneurysm, especially in the cavernous sinus; hemorrhage, particularly when the meninges at the base are torn; hydrocephalus of children; and thrombosis of the sinuses. Papillitis when due to the above causes is usually bilateral.

Cerebral tumors and *hydrocephalus* produce an increase of intracranial pressure, and the cerebrospinal liquid finds its way between the sheaths of the optic nerves, causing an edema of the papilla, known as choked disk. Inflammatory diseases at the base of the brain may reach the papilla by simple extension along the nerve.

The *orbital causes* are tumors, orbital cellulitis of whatever cause, and pressure by the bony walls of the optic foramen.

The *systematic diseases* which produce papillitis are the acute infectious diseases, syphilis, diabetes, anemia, chlorosis, and lead poisoning. Uterine affections, diseases of the other eye, chorea, or myelitis may be the cause of a papillitis. As a consequence of rheumatism or predisposition thereto, papillitis has been known to occur.

Prognosis.—Prognosis of a papillitis depends upon the intensity of the inflammation, but is never good. The damage to vision varies with the degree of atrophy which follows the inflammation.

Treatment.—Treatment of papillitis consists in correcting the cause, if possible, and in rest of the eyes and the use of diaphoretics and alteratives.

Retrobulbar Neuritis.—**Symptoms.**—This affects the orbital portion of the optic nerve, and may be either *acute* or *chronic*. The papilla does not show any characteristic change, except in the later stages, when atrophy may occur. The vision is impaired, and very often there is a central scotoma.

An **acute retrobulbar neuritis** is usually due to a cold, and is best *treated* by rest of the eyes; and in the early stages by the use of diaphoretics, as pilocarpine and the salicylates. The *prognosis* is usually good.

Chronic retrobulbar neuritis is usually due to tobacco and alcohol poisoning; but may occur in consequence of poisoning by stramonium, lead, sulphur, chloral, and the iodides; and occasionally complicates diabetes.

The visual field is contracted, and a central scotoma, first for green and red only and later absolute, is demonstrable upon perimetric examination. Chronic retrobulbar neuritis begins in middle age of men, who are more subject to the influences of various poisons than women.

Treatment.—The treatment consists in complete abstinence from tobacco and alcohol, or removal from the poisonous influence, and the use of strychnine, which may be used hypodermically, increasing the dose to tolerance, or administered internally in the form of a nux vomica preparation.

The *result of the treatment* is good in recent cases; not so good when considerable atrophy has already occurred.

Atrophy.—**Varieties.**—Atrophy (Fig. 24) of the optic nerve presents three pictures, and is accordingly classified as simple, postpapillitic, or retinitic.

Etiology.—*Simple atrophy* may be due: (1) to descending degeneration of the nerve fibers, caused by cerebral diseases which compress the optic nerve or tract; (2) to gray degeneration of the fibers in consequence of cerebral or spinal diseases; (3) to an acute or chronic retrobulbar neuritis; (4) to an embolus of the arteria centralis retinæ; (5) to various forms of retinitis; (6) to injuries affecting the continuity of the nerve; and (7) to exposure to cold, insufficient food, excesses, or mental emotion.

A *postpapillitic atrophy* follows a papillitis, and is accordingly due to the cause of the papillitis.

Retinitic atrophy is likewise due to the causes of retinitis, retinitis pigmentosa, and choroidoretinitis specifica, with which it frequently occurs.

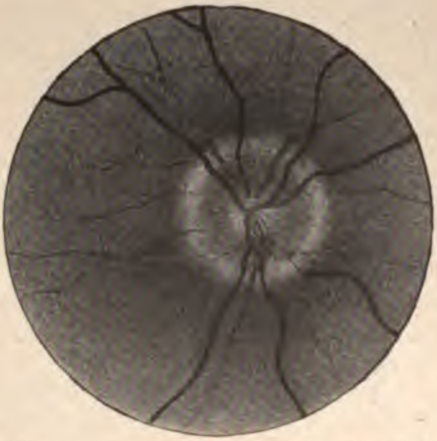

FIG. 24.—Ophthalmoscopic appearance of disk in atrophy following hemorrhage. (Jäger.)

The **atrophic excavation** which occurs in *simple atrophy* permits a view of the lamina cribrosa which gives the disk the stippled appearance. The lamina cribrosa is not pushed back in the atrophic excavation as it is in glaucomatous cupping. The papilla being swollen during a *papillitis*, never atrophies to the extent of producing an excavation, and this is the reason the physiologic cup is filled in.

Prognosis.—The prognosis of atrophy of the optic nerve is unfavorable, yet postpapillitic or retinitic atrophy may not become progressive.

The *appearance of the fundus* of the three forms of atrophy can be understood from the following scheme:

	Simple atrophy.	Postpapillitic atrophy.	Retinitic atrophy.
Color of disk	Bluish or greenish white and stippled	Hazy, bluish or grayish white and uniform	Dirty yellow or dark reddish gray.
Margin of disk	Very distinct and regular	Not more distinct than normal and blurred in places; often irregular	Blurred.
Surface of disk	Slightly concave: physiological cup not filled in	Flat; physiologic excavation filled in	At times concave.
Larger retinal vessels	Normal or nearly normal in size	Diminished in size, the arteries in particular; white lines, due to thickening of their walls, are seen along the vessels in places	Very considerably diminished in size or absent.

Treatment.—The treatment should be instituted according to the cause. Strychnine, iodides, and mercury may be tried, but with little prospect of success.

FUNCTIONAL DISEASES OF THE EYE.

Amblyopia.—Amblyopia is a term which expresses defective vision not due to refractive errors. It may be congenital; or occur from disuse of the eye.

Congenital Amblyopia.—Congenital amblyopia may complicate various congenital anomalies of the eye, as high degrees of astigmatism or hypermetropia, coloboma of the iris, choroid or ciliary body, hydrophthalmos, and microphthalmos. It is usually unilateral, and may not be noticed for many years.

Amblyopia ex Anopsia.—Amblyopia ex anopsia implies defective vision from disuse of the eye.

The disuse *may be due* to opacities of the cornea or lens which were caused in infancy. After development of the eye, which is completed in adult life, disuse of the organ is not likely to produce amblyopia. Strabismus occurring during infancy often leads to amblyopia which may affect half or all of the retina.

In some cases of *convergent strabismus*, the objects in the temporal half of the field of the squinting eye are seen; whereas those in the nasal half are not, from regional exclusion, which favors single vision.

Treatment.—The treatment for amblyopia ex anopsia consists in use of the eye, either by bandaging the other or by performing an operation to remove a cataractous lens. Discission may be done in infancy after three months of life, and the sooner it is done the better will be the result.

Hysterical Amblyopia.—Hysterical amblyopia is frequently observed in young, hysterical female subjects, and is characterized by an absence of intra-ocular changes together with impairment of vision. *Perimetric examination* shows a concentric contraction of the visual field, which becomes smaller the longer the patient is examined, and an overlapping of the color fields. The *prognosis* is good in most cases. The *treatment* consists in the use of tonics, electricity, and suggestion, which sometimes relieve all symptoms in a short time.

Amblyopia sometimes occurs in consequence of *slight cerebral changes* involving the optic tract. The more serious cerebral diseases usually produce changes of the optic nerve, as papillitis or atrophy.

Asthenopia.—Asthenopia or inability to use the eyes, sometimes complicates neurasthenia and hysteria. Use of the eyes causes pain or fogginess of vision, which prevent the patient from using them. Refractive errors and muscular asthenopia are not present in hysterical asthenopia. Electricity and suggestion have been found useful in some cases, but often very little is accomplished by any treatment excepting rest.

Amaurosis.—Amaurosis implies absolute blindness which is not due to changes in the refractive media. It may be temporary or permanent. Uremic poisoning may produce temporary amaurosis; cerebral lesions, permanent blindness.

Amaurosis partialis fugax is a form of temporary blindness due to some central cause. An attack often begins with headache and vomiting, and after objects have for a short time appeared blurred, vibrating, and sparkling, blindness supervenes. Vision returns soon in most cases and the sight is not damaged.

Achromatopsia.—Achromatopsia or color-blindness, may be congenital or acquired.

When *congenital*, it may be partial, in which case only certain colors cannot be distinguished; or complete, when all objects appear gray. Color-blindness for *red* is the most common form of congenital color-blindness, and males are more subject to it than females,

The *detection of color-blindness* is of great importance in railway employees, who should be able to distinguish colors in order to interpret signals. In order to test the perception of colors, skeins of worsted of many shades of the primary colors are given the person to be examined, and he should be requested to put aside all skeins which to him appear blue, red, green, etc., as the case may be.

Acquired color-blindness is due to disease of the retina, optic nerves, or optic tracts, and never to disease of the media. The loss of perception of color is gradual, some colors being recognized longer than others.

Green, red, yellow, and blue fail to be recognized in the order given in disease of the optic nerve.

Hemeralopia.—Hemeralopia is a symptom of many diseases, and implies that although the vision during the daytime is good, at night it is bad, or even *nil*. Peripheral opacities of the cornea or lens in bright light do not interfere with the course of the rays of light through the contracted pupil, but interfere seriously with it in the dark, when the pupil is dilated.

When the peripheral portion of the retina is diseased, the central portion is sufficient to give good vision during the daytime, the pupil being contracted; but at night vision suffers to the extent of the retinal disease.

Hemeralopia is sometimes observed in individuals whose physical condition is below par because of insufficient or improper food. It not infrequently occurs in jaundice, chronic alcoholism, or pregnancy. The *prognosis* is good, and the success of the *treatment* depends upon the restoration of health.

Nyctalopia.—Nyctalopia implies good vision at night, when the pupil is dilated, and bad during the day, when the pupil is contracted. The cornea, or lens, has a central opacity, or the retina is centrally diseased when there is nyctalopia. In chronic retrobulbar neuritis nyctalopia is a prominent symptom.

THE MUSCLES OF THE EYEBALL.

ANATOMY.

The eye has two kinds of muscles, extrinsic and intrinsic.

The **extrinsic** muscles are six in number, and their function is to change the direction of the eye so that objects may be brought into the field of vision. These are the superior, inferior, external, and internal recti, and the superior and inferior oblique (Fig. 25).

The **intrinsic muscles** are the ciliary and sphincter pupillæ, which are concerned respectively in accommodation and contraction of the pupil. These, like the superior, internal, and inferior recti, and the inferior oblique, are supplied by the third nerve; and, owing to the intimate relation in the floor of the Sylvian aqueduct of the nuclei of the nerve fibers to the ciliary muscle, the iris, and three extrinsic muscles, convergence, accommodation, and contraction of the pupil are associated acts.

Axes, Planes, and Meridians.—The *sagittal*, *optic*, or *antero-posterior axis* of the bulb is a line extending from the centre of the cornea to the posterior pole. The *visual axis* is a line which extends from the yellow spot to the cornea in the direction of the object looked at. The visual and the antero-posterior axes practically coincide.

The centre of motion of the bulb is a point on the anteroposterior axis one line behind its centre.

When a horizontal plane passes through the anteroposterior diameter of the bulb, it forms at its intersection with the external surface of the bulb the *horizontal meridian*.

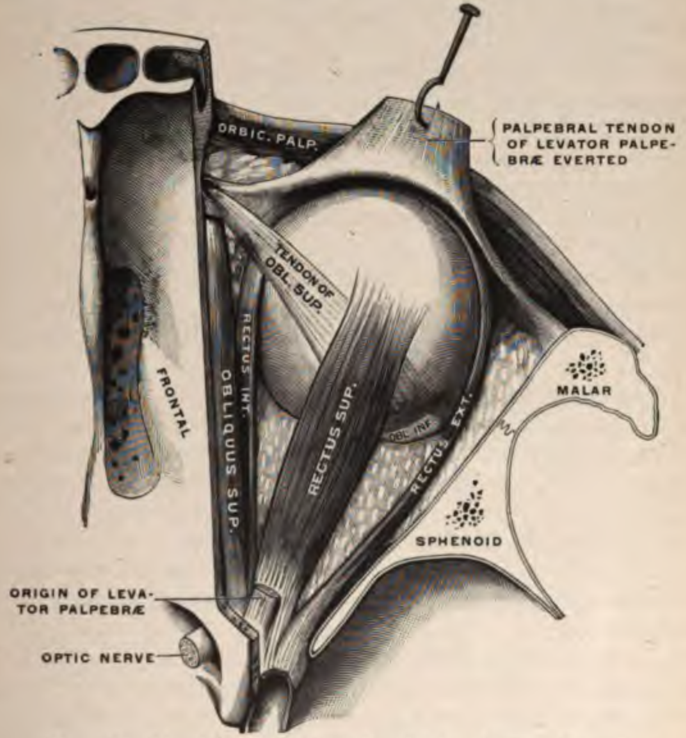

FIG. 25.—Muscles of right eye, viewed from above. (Testut.)

A vertical plane passing through the anteroposterior diameter forms at its intersection with the external surface of the bulb the *vertical meridian.*

The *equatorial meridian* represents the intersection of a plane passing through the centre of motion and perpendicular to the anteroposterior axis with the external surface of the bulb.

The horizontal, or *frontal, axis* of the bulb is a line passing through the horizontal meridian at right angles to the anteroposterior axis at the centre of motion.

The *vertical axis* of the bulb is a line passing through the vertical meridian at right angles to the anteroposterior axis at the centre of motion.

A line connecting the centre of motion of each eye forms the *base line*.

A plane passing through the centre of the base line and perpendicular to it is called the *median plane*.

The *visual plane* passes through the base line and the visual axis or lines.

The plane passed through the centres of origin and insertion of an extrinsic muscle and the centre of motion of the bulb, is called a *muscle plane*.

Each extrinsic muscle when functioning alone moves the eye around the so-called *axis of turning*, which is perpendicular to the muscle plane and passes through the centre of motion.

Positions of the Eyeball.—An eye is said to be in the *primary position* when the muscles are perfectly balanced and the visual lines are in a horizontal plane and parallel to the median plane; other positions are *secondary*. In studying the action of a muscle the change of position of the centre of the cornea, either elevation or depression, and adduction or abduction, should be noted; also the movement of the point of intersection of the equatorial and vertical meridians on the top of the eye.

When this point moves toward the nose, it implies *nasal*, or + *wheel rotation;* and when toward the temple, *temporal*, or — *wheel rotation*.

The **internal rectus** arises from the tendon of Zinn and the inner margin of the optic foramen, and terminates in an aponeurosis, four and one-half lines broad, which is inserted into the sclerotic coat about three lines from the internal margin of the cornea. It is the broadest of the recti ocular muscles, and pulls the cornea directly inward (adduction) and there is no wheel rotation.

The **external rectus** is the longest of the recti muscles and has two heads of origin: The upper one arises from the outer margin of the optic foramen, and the lower partly from the tendon of Zinn and partly from a pointed process of bone on the lower margin of the sphenoidal fissure. It terminates in an aponeurosis three and a half lines broad, which is inserted into the sclerotic coat about four lines from the outer margin of the cornea; and, like the internal rectus, on a level with the centre of it.

This muscle pulls the cornea directly outward (abduction), and there is no wheel rotation since the vertical axis remains unchanged.

The **movements** of the centre of the cornea are in the line of the horizontal meridian during the action of either the *external* or the *internal rectus*, and hence there is neither elevation nor depression of the eye.

The action of either of the *other four* extrinsic muscles causes either adduction or abduction, elevation or depression, and nasal or temporal wheel rotation: their action, therefore, produces *compound motion*.

The **superior rectus** is the thinnest and narrowest of the recti muscles, and arises from the upper margin of the optic foramen and is inserted obliquely by a tendinous expansion into the sclerotic between three and four lines from the superior margin of the cornea.

The superior rectus elevates and adducts the centre of the cornea, and nasal wheel rotation takes place.

The **inferior rectus** has a common origin with the internal rectus, and is, like the superior rectus, obliquely inserted into the sclerotic, the inner edge two and a half, and the outer three and a half, lines from the lower margin of the cornea.

The inferior rectus depresses and adducts the centre of the cornea, and the wheel rotation is temporal.

The **superior oblique** arises about a line above the inner margin of the optic foramen and passes forward, upward, and inward to the upper inner angle of the orbit to a ring of fibrocartilaginous tissue, through which it continues, and is then reflected backward, outward, and downward beneath the superior rectus to be inserted by a fan-shaped aponeurosis into the sclera between the superior and external recti muscles, and midway between the margin of the cornea and the entrance of the optic nerve.

The superior oblique causes principally nasal wheel rotation and the centre of the cornea is depressed and abducted.

The **inferior oblique** arises from a depression in the orbital plate of the superior maxillary bone, external to the orifice of the nasal duct, and, passing outward, backward, and upward, first beneath the inferior rectus and then between the external rectus and eyeball, is inserted by a fan-shaped aponeurosis into the sclerotic, near the insertion of the superior oblique. The anterior edge of the insertion is five lines from the cornea, and the posterior edge two lines from the entrance of the optic nerve.

The inferior oblique more particularly causes temporal wheel rotation, and the centre of the cornea is elevated and abducted.

Summary of Actions:

MUSCLE.	ACTION.
Internal rectus,	Adduction of centre of cornea.
External rectus,	Abduction of centre of cornea.
Superior rectus,	Elevation of centre of cornea. Adduction of centre of cornea. Nasal wheel rotation. Highest point of the vertical meridian moves toward the nose.
Inferior rectus,	Depression. Adduction. Temporal wheel rotation. Highest point of the vertical meridian moves toward the temple.
Superior oblique,	Depression. Abduction. Nasal wheel rotation.
Inferior oblique,	Elevation. Abduction. Temporal wheel rotation.

Superior rectus and inferior oblique—Elevation.
Inferior rectus and superior oblique—Depression.
Superior and internal recti and inferior oblique—Elevation and adduction.
Superior and external recti and inferior oblique—Elevation and abduction.
Inferior and internal recti and superior oblique—Depression and adduction.
Inferior and external recti and superior oblique—Depression and abduction.

The extrinsic ocular muscles receive their **blood supply** from the ophthalmic artery.

The **sensory nerves** are from the trigeminus, mostly from the first division, but also from the orbital branch of the second division.

The **motor nerves** are the third, fourth, and sixth. The *third* supplies the superior, inferior, internal rectus, and inferior oblique; also the intrinsic muscles, the ciliary and sphincter pupillæ; and, finally, the levator palpebræ superioris, and explains the existence of ptosis, paralysis of accommodation, dilatation of the pupil, and divergence (paralysis of the adductors).

The *fourth*, or pathetic, nerve supplies the superior oblique; and the *sixth*, or abducens, the external rectus.

ANOMALIES OF THE MOTOR APPARATUS.

The **anomalies** of the motor apparatus of the eyes include *heterophoria; strabismus*, or *heterotropia*, which may be paralytic or non-paralytic; and *nystagmus*.

When the extrinsic, or extra-ocular, muscles are in perfect equilibrium and the visual lines are kept parallel with little effort, the normal condition, or *orthophoria*, exists.

When, however, a tendency to deviation of the eyeball occurs, and this can be overcome by special effort, *heterophoria* is the name given to the condition.

When the tendency to deviation cannot be overcome and the visual lines are not directed to the same point of regard, *strabismus*, or *heterotropia*, results.

Heterophoria.—Heterophorias are divided according to the tendency of the visual lines as follows: *esophoria*, *exophoria*, *hyperphoria*, *hyperesophoria*, and *hyperexophoria*.

Esophoria.—Esophoria occurs when the visual lines tend to turn *inward*, owing to the superiority of action of the corresponding muscles.

To demonstrate the presence of an esophoria, a card should be put before one eye and the patient told to observe a fixed point with the other. On removal of the card it will be noticed that this eye makes a slight excursion outward. While the card covered the eye it was slightly turned inward. After removal of the card it is necessary for the eye to make an excursion outward in order to bring the visual lines upon the same point of regard.

The *degree* of esophoria can be determined by seating the patient at least twenty feet from a source of light (a candle answers very well) and placing horizontally before one eye a Maddox rod and before the other a prism, base out, which will bring the vertical strip or bar of light through the light.

The degree of the prism represents the degree of esophoria. It will be noted that in esophoria, the Maddox rod being before the patient's right eye, the strip of light will be to the patient's right of the light.

Convergence and accommodation must not be used in making this test to determine the muscular equilibrium, and hence a distance of at least twenty feet between the patient and light is essential to make the test of value.

Exophoria.—Exophoria occurs when the visual lines tend to diverge.

When one eye is covered with a card there is a tendency to divergence; after removal of the card the eye makes an excursion inward.

With the aid of a Maddox rod horizontally placed, the presence of exophoria can be determined, the strip of light being toward the nasal side of the eye before which the rod is placed.

The *degree* of the prism, with the base in, which brings the bar of light and the light in a line represents the degree of exophoria.

Hyperphoria.—Hyperphoria is that condition of the ocular muscles which produces a tendency of the visual line to pass above that of the other eye. When the visual line of the right eye is above that of the left, right hyperphoria exists; when the visual line of the left eye is above that of the right, then there is left hyperphoria. The superior rectus of the right eye causes more elevation of the cornea than the superior rectus of the other in right hyperphoria. In left hyperphoria the condition is reversed.

The Maddox rod vertically placed before the right eye shows the existence of right hyperphoria when the bar of light is below the light, and left hyperphoria when above.

Hyperesophoria.—Hyperesophoria is the condition in which there is the compound tendency of the visual line to rise above the other and also to pass inward.

Hyperexophoria.—In hyperexophoria the visual line has a tendency to pass outward and to rise above the other.

Etiology.—The causes of the heterophorias may be *organic* or *functional*.

The *organic causes* are muscular weakness, which may be due to *smallness* of the muscle, the point of insertion, and the size of the eyeball. Weakness may also occur after exhausting diseases or previous paralysis.

The *functional causes* depend upon the association of convergence and accommodation. During the various degrees of accommodation, convergence in normal eyes is so regulated that the muscular equilibrium is perfect. Eyes with refractive errors, as hypermetropia and myopia, demand more or less accommodative effort than the normal eye. The myopic eye requires less, the hypermetropic eye more, accommodation than the normal eye, all fixing for the same distance.

In myopia, less demand being made upon the accommodation, less is required of the internal recti; and hence a weakness, or insufficiency, of the internal recti may develop.

In hypermetropia more accommodative effort is required to see well near by than in emmetropia, and hence the demands on the internal recti are greater than are normally required, resulting in increase of function, development, and strength. The internal recti would then be relatively stronger than the external.

Insufficiency, or weakness of the interni manifests itself, when accommodative effort is prolonged for near use of the eyes by indistinct vision, diplopia, and headache. This constitutes *muscular asthenopia*, which is characteristic in that the symptoms subside when one eye is closed and no convergence is necessary.

Treatment.—The treatment of the heterophorias depends upon the amount of trouble they occasion and the cause. When no disagreeable symptoms, either ocular or reflex, occur, they may be disregarded. Refractive errors should in every case be corrected, and special attention should be given to the correction of astigmatism when it is present. Prisms may be employed in some cases when the degree of the heterophoria does not exceed 6 degrees. A prism of 3 degrees could be used before each eye to get the effect of 6 degrees, for the weight of the glass and the dispersion of light militate against the use of strong prisms.

In *esophoria* the proper prism with the base out may give relief; in *exophoria* the prism should have the base in; and in *hyperphoria* the base should be down.

It will be observed that the base is always directed to the side of the weaker muscle.

In order to develop the weaker muscles they may be exercised by the use of prisms so placed that an additional effort is imposed upon the muscle to be exercised. These prisms may be mounted in a so-called *grab front*, and used by the patient by attaching it to his spectacles (of which most patients have need) several times a day for some minutes.

In some cases the only treatment is a *tenotomy* of the stronger opposing muscle. The tenotomy may be partial or complete, according to the effect desired. The partial tenotomy, involving only a part of the tendinous expansion of the muscle, may be graduated according to the exigency of the case. The effect of such a tenotomy should be watched, and if necessary repeated until the desired result is obtained.

Low degrees of heterophoria when subject to variations should not be operated upon.

It should be kept in mind that a hyperphoria of *two degrees* may cause great distress when an esophoria or exophoria of such a degree may not be noticed, the excursions of the eye from side to side being much greater in amplitude than those up and down.

Strabismus.—Strabismus is classified as *paralytic* or *non-paralytic*.

Movements of the Eye.—In *non-paralytic strabismus* the movement of one eye is always accompanied with a similar movement of the other, and hence this form is called *concomitant strabismus*.

The movement of the eyeball in *paralytic strabismus* is entirely deficient to the extent of the muscle or muscles paralyzed, whereas the movement of the eyeball in concomitant squint is the same in both eyes.

Deviations.—The *primary deviation of concomitant squinting eyes* is the difference between the position of the squinting eye when the other is fixing and the position of the same eye when it is fixing. *Secondary deviation* is the difference between the position of an eye when fixing and its position when the other eye is fixing. In concomitant squint the primary and secondary deviations are equal.

In a *paralytic squint* the *primary deviation* represents the angle between the line of vision and the line from the object to the nodal point of the paralyzed eye. If a card be placed before the sound eye, it will deviate more than the paralyzed eye when the sound eye was fixing; this is the secondary deviation. In non-paralytic squint the primary and secondary deviations are equal; in paralytic squint the secondary deviation exceeds the primary.

Symptoms.—The symptoms of a paralytic squint are: loss of motility; diplopia; vertigo; compensatory position of the head; the secondary deviation is greater than the primary; and there is false projection.

The *loss of motility* depends upon the muscles paralyzed. When the third nerve is affected, all the extra-ocular muscles except the external rectus and superior oblique may be paralyzed. The eye protrudes slightly, since three of the recti are lax; the eye is turned outward and downward; there is ptosis; the pupil is dilated and accommodation suspended. When all the extra-ocular and intra-ocular (ciliary muscle and sphincter pupillæ) muscles are paralyzed, the condition is called *ophthalmoplegia totalis;* when the extrinsic ocular muscles are paralyzed, *ophthalmoplegia externa;* and when the intrinsic (ciliary and sphincter pupillæ) muscles are affected, *ophthalmoplegia interna*.

When paralysis affects both eyes in such a way that associated movements are not possible, the condition is known as *conjugate paralysis*.

Diplopia.—*Binocular diplopia* occurs in the more recent cases, and is due to the fact that the visual axis of one eye deviates from the point of regard. *Homonymous diplopia* occurs when there is undue convergence of the eyes, and the right eye sees the image to the right side and the left eye the image to the left.

When the images are crossed, the image to the left being seen by the right eye and the image to the right by the left eye, the condition is called *heteronymous* or *crossed diplopia.* This occurs in divergence of the eyes.

Both forms occur in paralytic squint according to the kind, but evidently diplopia appears when only the eyes are moving in the direction requiring the function of the paralyzed muscle.

Vertigo and Compensatory Position of the Head.—The patient, in order to prevent vertigo and diplopia, *holds the head* in such a way that the affected muscle does not come into play. As a rule the head is turned in a direction opposite to that of the functioning muscle.

The *secondary deviation* is greater than the primary because the extra impulse to a paralyzed muscle of the diseased eye causes but slight response; whereas the similar impulse to the corresponding muscle of the other eye produces the full effect.

False projection occurs in an eye with a paralyzed muscle, the sound eye being covered when the function of that muscle is required to see an object. The patient believes that he sees the image at a point corresponding with the impulse to the paralyzed muscle.

In *recent paralyses* the diplopia, vertigo, and compensatory position of the head will be well marked; but in *old cases* the retina of the diseased eye learns to exclude the images, and these symptoms are not present. In old paralyses contracture of the antagonists may occur.

Etiology.—The etiology of paralyses of the ocular muscles may be due to intracranial or orbital lesions.

These are very often the result of syphilis; but cerebral diseases (particularly those at the base of the skull), tuberculosis, diabetes, diphtheria, injury, rheumatism, and exposure to cold are also causal.

Treatment.—The treatment of paralytic squint will depend upon the cause. When there is a syphilitic history, mercury and the iodides should be administered, and when there are no intra-ocular diseases salvarsan or neosalvarsan should be tried. In rheumatic paralyses the salicylates and diaphoretics are indicated. Use of the constant current is applicable to all recent cases. The paralyzed muscle may also be exercised with the aid of prisms. The employment of prisms is sufficient to relieve the diplopia and vertigo when the squint is of low degree; but an opaque plate before the paralyzed eye is necessary in bad cases. When contracture of an antagonistic muscle occurs, the condition may be remedied by a tenotomy of this muscle and an advancement of the paralyzed one. Complete paralyses are incurable.

Varieties of Non-paralytic or Concomitant Strabismus.—This is *convergent* when one eye turns inward; *divergent*, when one eye turns outward; *monolateral*, when the same eye always squints; and *alternating*, when sometimes one and sometimes the other eye squints. It may be *periodic* or *constant*.

Etiology of Convergent Strabismus.—It is essentially due to defect of the fusion faculty provoked by hypermetropia, anisometropia hyperphoria, esophoria, specific fevers, and violent mental disturbance. The influence of heredity has been established in 50 per cent. of cases. Opacities of the cornea or lens and intra-ocular disease may also be causative.

Convergent strabismus is very often associated with hypermetropia. This is due to the fact that hypermetropes require a great effort to accommodate for near vision, and because of the connection between convergence and accommodation sufficient accommodation requires a strong impulse toward convergence. Hypermetropia when associated with a visual disturbance which is the result of a congenital condition or postnatal disease is likely to lead to strabismus. When the eyes are first required for near vision—that is, between the age of three and six—strabismus most frequently develops. Very often the squint is periodic, being noticed only when the child fixes for near objects; later it may become constant.

Treatment.—The treatment of convergent strabismus should always begin with a correction of the refractive errors. Children should not be operated upon before six years of age, when they begin to attend school and require their eyes for near work. Children under six years should be refracted after a retinoscopic examination, and the full correction should be prescribed. It is also well to keep the fixing eye *only* under the influence of a cycloplegic (atropine) for several months. In order that the squinting eye may not become amblyopic from disuse, its employment should be forced by bandaging the other eye continuously for some months. Training the fusion sense, if begun very early, preferably between the second and fifth year, is of great value. Spontaneous cure sometimes takes place.

If after faithful trial of non-operative measures the strabismus does not diminish, a *tenotomy* of the internal rectus must be performed. When alternating strabismus is present, the eye with worse vision should be first operated, and if after one or two months the squint is still present, the internal rectus of the other eye should be tenotomized. An advancement of the external rectus together with tenotomy of the internal rectus is seldom required.

Divergent strabismus is very frequently associated with myopia. For near vision a myope needs little or no accommodation, and the impulse to the internal recti is accordingly small. As the myopic eye is often increased in size, its inward movement is also mechanically interfered with. When the convergence is insufficient for near vision, one eye turns out. Divergent strabismus begins in youth (children are very seldom myopic), and tends to increase with age. The treatment of myopic divergence is first of all the correction of the myopia and myopic astigmatism. The patient should wear glasses for both near and distant vision. An operation has frequently only a cosmetic effect, for vision is not often restored. The simple operation of tenotomy of the external rectus is seldom sufficient, an advancement of the internal rectus being also necessary. In some cases nothing helps.

Nystagmus.—Nystagmus is a condition of the eyeball in which rapid, lateral, vertical, or rotatory movements take place. It usually affects both eyes when the movements of whatever kind are simultaneous.

Nystagmus may be *due* to amblyopia which developed at an early age, congenital anomalies, great refractive errors, or retinitis pigmentosa. It also occurs with cerebral diseases, conditions of irritation of the labyrinth of the ear due to an otitis or disturbance of equilibrium and develops in coal-miners because of the position in which they are sometimes obliged to work. Coal-miners' and labyrinthine nystagmus may cease when it is possible to remove the cause. Other forms of nystagmus are not amenable to *treatment*, and the correction of coexisting refractive errors will be found unsatisfactory.

REFRACTIVE ERRORS.

Varieties.—As regards its refractive condition, the eye is either emmetropic (normal) or ametropic (abnormal). In an *emmetropic eye* parallel rays from distant objects (twenty feet and more) are focussed on the retina without any accommodative effort.

When the rays do not focus on the retina, the conditions being the same, the eye is *ametropic*.

When parallel rays passing through all meridians of the cornea focus in front of the retina, the ciliary muscle being relaxed, the condition is called *myopia*.

When the rays focus behind the retina, the condition is one of *hypermetropia*.

When the parallel rays passing through one meridian of the cornea focus on the retina and others, passing through a meridian at right angles to the first, focus in front of the retina, the condition is called *simple myopic astigmatism;* when behind the retina, *simple hyperopic astigmatism* is present.

When parallel rays passing through two meridians at right angles to each other focus at different points in front of the retina, *compound myopic astigmatism* is present.

When the rays focus at different points behind the retina, the condition is one of *compound hyperopic astigmatism*.

When the refraction of the two eyes is different, the condition is called *anisometropia*.

Myopia.—Etiology.—Myopia may be due to an increase of the refractive power of the eye or to an elongation of its axis. An increase of corneal or lenticular curvature, an increase of density of the lens, and spasm of the ciliary muscle, whereby the convexity of the lens remains at its greatest point, are the causes of abnormally great refracting power of the eye. *Axial myopia* is due to distention of the sclera, and when the bulging occurs at the posterior part of the eye it forms the so-called *posterior staphyloma*. There may also be a displacement of the inner or the outer layer of the sclerotic, producing a change in shape of the discal end of the intervaginal space (Fig. 26).

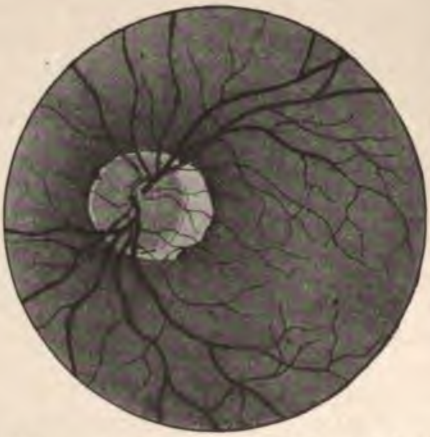

Fig. 26.—Ophthalmoscopic view of change in shape of discal end of intervaginal space. (Jäger.)

In order to determine the degree of myopia, the patient should be seated twenty feet from the test-types, and concave glasses, beginning with the weaker and gradually increasing, should be put before the eye to be examined: the weakest concave glass which gives the best vision represents the degree of myopia. The degree of myopia varies from a very low amount to twenty, or rarely, thirty dioptres.

In prescribing glasses for myopia of two or even three dioptres, glasses are not necessary for near work, but are usually very desirable for distant vision. When astigmatism is also present, its correction is often of advantage for near work. When the myopia is less than seven dioptres, one pair of glasses not quite correcting the myopia suffices for near and distant vision when the accommodation is good. If not good, two pairs of glasses should be prescribed, one quite correcting the myopia for distance and a weaker one for near work. When the myopia exceeds seven dioptres, two pairs of glasses will generally be required.

Hypermetropia.—**Etiology.**—Hypermetropia may be due to diminished refractive power of the media or to a shortening of the axis of the eye. A flattening of the cornea and diminished density or absence of the lens diminish the refractive power of the eye. Axial hypermetropia may be congenital or due to protrusion of the retina.

When a hyperope has not the necessary accommodative power to see distinctly at twenty feet, the strongest convex glass necessary to produce distinct vision represents the degree of *manifest hypermetropia.*

The convex glass necessary to give distinct vision at twenty feet when the ciliary muscle is paralyzed, represents the *total hypermetropia.*

The difference between the total and manifest hypermetropia is the amount concealed by the accommodation, and is called the *latent hypermetropia.*

When the power of accommodation of a hyperopic eye is reduced to *nil,* or is insufficient to bring parallel rays to a focus on the retina, the condition is called *absolute hypermetropia.*

The *amplitude of accommodation,* which is the difference of refractive power of the eye when at rest and when accomodation is exerted to the utmost, is the same in a hypermetropic and an emmetropic eye; but the near point of the former is at a greater distance from the eye than that of the latter. In consequence of the extra effort of accommodation necessary for near vision, the ciliary muscle becomes tired, and this condition is known as *accommodative asthenopia,* not to be confounded with *muscular* and *nervous asthenopias.*

Treatment.—The treatment of hypermetropia with convex glasses is very satisfactory. A hypermetropia less than 1 D. after use of atropine can usually be disregarded. When it exceeds 1 D., as a rule, a glass should be prescribed weaker by 0.25 D. for every dioptre of hypermetropia. In children more may be deducted with advantage; and when the degree of hypermetropia in adults exceeds 3 or 4 D., the same exception to the rule applies.

Presbyopia.—**Etiology.**—Presbyopia—"old sight"—is the result of a senile change to which every eye is subject about the age of forty years. It is due to the impaired elasticity of the lens, in consequence of which its convexity is insufficient for near work.

Treatment of Presbyopia in Emmetropic Eyes.—The aid of convex glasses supplies the want of elasticity which increases with age up to a certain point. As the inherent elasticity of the lens and its consequent convexity diminish, so the near point at which perfect vision is possible recedes from the eye. When presbyopia begins, this point is 22 cm. The table below gives the distance of the near point from the eye, and the strength of the convex glass necessary to bring this point to 22 cm. in emmetropic eyes for the various ages.

PRESBYOPIA TABLE FOR EMMETROPIC EYES.

	Distance of *p*.		Pr. expressed by the lens necessary to bring the *p* to 22 cm. or 9″.	
	Cm.	Inches.	Dioptres.	Paris inch scale.
40	22	9	0	0
45	28	11	+1	$+\frac{1}{35}$
50	43	17	2	$\frac{1}{18}$
55	67	27	3	$\frac{1}{12}$
60	200	72	4	$\frac{1}{9}$
65	Infinity.		4.5	$\frac{1}{8}$
70	Acquired H = 1 D.		5.5	$\frac{1}{6\frac{1}{2}}$
75	" H = 1.5 D.		6	$\frac{1}{6}$
80	" H = 2.5 D.		7	$\frac{1}{5}$

The above table applies only to emmetropic eyes; hence it is necessary to consider the *treatment when the eyes are ame-*

tropic. The visual acuity in every case for twenty feet should be determined, and the glass found which gives the best vision. If the patient is a *hyperope*, the glass representing the manifest hypermetropia should be added to the convex glass recommended for an emmetropic eye of an individual of the same age; if he be a *myope*, the amount should be subtracted. In every case the correction should be tried with test-types for near vision, and changes, if necessary, be made.

Astigmatism.—Varieties.—Astigmatism is either *regular* or *irregular*. When the curvature of the refracting media is regular in every meridian, however different the curvature of any two meridians may be, the astigmatism is regular; when the curvature in a meridian is not regular—that is, not the same throughout—the rays passing through it do not focus at a point, and the astigmatism is irregular. Regular astigmatism can be corrected by proper cylindrical glasses, but it is not possible to correct the irregular form.

Regular Astigmatism.—Regular astigmatism may be corneal or lenticular, or both. The amount of corneal astigmatism and its axis can be quickly and objectively determined with the aid of an ophthalmometer. Retinoscopy affords a method of determining the amount of a regular astigmatism of whatever kind, and approximately the axis. Subjectively, the astigmatism can be determined with the aid of cylindrical lenses and an astigmatic chart. The axis of the cylinder which is necessary to make all lines of the chart equally distinct will be at right angles to the meridian which requires correction, and the strength of the cylinder is the degree of the astigmatism. The amount of astigmatism represents the difference between the greatest and least curvatures in two meridians, called the principal meridians, which are at right angles to each other. Very frequently one meridian is vertical, the other horizontal. Generally the vertical meridian has the greater curvature and when this is the case, the astigmatism is said to be "with the rule;" when, however, the horizontal has the greater curvature, the astigmatism is "*against the rule.*"

When the refraction of one meridian is emmetropic and the other hypermetropic, the condition is one of *simple hyperopic* moscopic and possibly retinoscopic examination will disclose the refractive error.

For the examination of *presbyopes* a cycloplegic is not necessary, and may be dangerous in very rare cases from increase of intra-ocular pressure.

In correcting the **heterophorias** it is well to ascertain the adduction, abduction, and circumduction at twenty feet. Abduction is determined by placing before a patient's eye the strongest prism, base *in*, which does not produce diplopia not to be overcome; adduction, by placing the strongest prism, base *out;* and circumduction, base *down*. The power necessary to overcome a prism, base placed *down* before the right eye, represents the right circumduction; before the left eye, left circumduction. Normally, abduction is about 7 degrees; adduction, from 30 to 40 degrees, according to practice; and circumduction, 2 degrees. A case of heterophoria should not be *operated* upon until the effect of spherical cylindrical, and prismatic glasses has been tested.

When prisms give relief after spherical and cylindrical glasses have failed, the patient may be operated upon if, for esophoria or exophoria, a prism of at most 6 degrees does not correct the trouble.

A hyperphoria of 2 degrees is often relieved by a graduated tenotomy. Heterophorias of high degree require surgical interference.

OPERATIONS.

Ophthalmic operations represent a comparatively small amount of eye work and the student will have but little opportunity to do the operations on the human subject. Moreover, it is absolutely unfair to patients to subject them to operations which require such perfect technic and great experience for their proper performance until by practice sufficient skill has been developed. No beginner learns to perform operations by watching even a most skilful operator; it is necessary to do them. Lid operations may be practised on the human cadaver as soon as possible after death, muscle operations on sheep's heads with the eyes *in situ* and the intra-ocular operations on the eyes of kittens four to six weeks old. After enucleation the kittens' eyes may be secured in a phantom or on one side of a cigar box, into which a small hole (1 cm.) has been drilled, and anchored to a tack on the inside. At very little expense, the student can obtain the material sufficient to do as many operations as falls to the lot of most oculists in a life-time and the skill and confidence acquired in this practice will be reflected in every instrumental manipulation made on or about the patient's eye.

LID OPERATIONS.

Trichiasis.—Trichiasis may be relieved by ablation, transplantation, elevation of the bed of hair follicles, or by correcting the position of the tarsus:

For *ablation* the lid is supported on a horn plate, and with a small scalpel an incision about 3 mm. deep is made through the free border of the lid between the orifices of the Meibomian glands and the cilia the whole length of the lid; the flap containing the cilia is then excised.

For *transplantation*, a horizontal fold of skin is excised and an incision as for ablation, but slightly deeper, is made. The wound of the integument is then sutured, whereby the bed of follicles is drawn up in proportion to the size of the excised skin. The excised skin is trimmed to fit the exposed surface below and grafted.

The bed of hair follicles can be drawn up by *Hotz's method:* An incision is made across the lower margin of the tarsal cartilage of the upper lid and the exposed part of the orbicularis is excised. Three or four sutures are used to close the wound in such a way that the tarsal cartilage is included in each suture, giving the skin firm attachment to the cartilage.

The position of the tarsus may be corrected, *Snellen's operation*, by excising from it a wedge-shaped piece after the skin has been incised and the muscle excised, as is done in the Hotz operation. The lower border of the lid is everted and kept in this position by several sutures. Each suture is armed with two needles, one of which is introduced into the upper border of the tarsus to give firm support; and, after the suture has been drawn through the cartilage, both needles are pushed through the integument of the lower lip of the wound at a short distance from each other. The needles are then removed, and each suture is tied upon a bead and finally attached to the forehead with plaster.

Spasmodic Entropion.—Spasmodic entropion of the lower lid occurs in old people when there is relaxation of the superabundant skin and contraction of the orbicularis muscle. To relieve this condition a horizontal fold of skin equal to the breadth of the lid should be excised, and also the part of the orbicularis exposed by the wound. The instruments necessary are a horn plate, scalpel, scissors, and forceps.

Organic Entropion.—Organic entropion frequently causes trichiasis, and the operations suggested for the latter are frequently applicable to the former.

Ectropion of the lower lid of old people due to relaxation of the tissues may be improved by removing a V-shaped piece of integument and bringing the edges of the wound in apposition. When the ectropion is slight with little loss of tissue, a V-shaped incision involving the scar producing it is made, and the flap dissected up until the lid is no longer everted. When the lower edges of the wound are sutured, the V becomes a Y. The operation is completed by suturing the arms of the Y.

When the ectropion is extreme and there is considerable loss of tissue, a **blepharoplasty** is necessary. To perform this a flap may be transplanted from the neighboring skin with a pedicle; the adjacent skin may be pushed into the gap; or small dermic or epidermic grafts may be used.

Canthoplasty.—A canthoplasty is an operation designed to lengthen the palpebral fissure at the outer canthus. The lids are separated by a speculum or with the fingers of the left hand, and the canthus cut with a pair of strong scissors. The skin, muscle, conjunctiva, and external palpebral ligament are divided by the cut. The ocular conjunctiva near the incision is dissected up and included in the sutures, of which three are necessary—one at each margin of the wound and one at the angle.

Tarsorrhaphy.—A tarsorrhaphy is an operation designed to shorten the palpebral fissure. This may be accomplished by excising the lid margins of both lids, including the roots of the cilia near the outer canthus, and stitching the denuded surfaces together.

Operations for Ptosis.—Operations for ptosis, particularly the congenital form, and any other which has not been successfully treated by other methods, have been devised by Wilder and Panas.

To perform *Wilder's operation,* general anesthesia is necessary. It is done as follows: After the eyebrow has been shaved, an incision about one and a half inches in length is made just above the margin of the orbit and parallel with it down to the periosteum. The lower lip of the wound is then retracted, and the integument and muscle are dissected from the underlying tarso-orbital fascia until the tarsus is partially exposed. Two sutures of fine sterilized silk or catgut, each of which armed with needles at both ends, are then required. The first needle of one suture is pushed through the upper part of the tarsus about the middle of the outer half, in order to secure a firm hold for the suture, and is then passed through the tarso-orbital fascia vertically upward toward the margin of the orbit in such a way that a purse-string suture results. Finally, the needle should include the muscle and connective tissue of the upper lip of the wound.

The second needle is introduced into the tarsal cartilage a little to the nasal side of the point where the first needle was introduced, and passed through the tarso-orbital fascia in the same manner as was the first needle, and after it has included the muscle and connective tissue of the upper lip of the wound, both ends of the suture may be drawn up to the extent necessary to relieve the ptosis. The second suture is introduced in the same way as the first, about the middle of the inner half of the tarsal cartilage, and then both sutures are tied after sufficient traction has been made.

After cleansing the wound, the operation is finished by coapting the edges of the primary incision with the necessary sutures and applying a firm bandage. These sutures should be removed in five or six days, but the deep sutures allowed to remain. Removal of the deep sutures because of suppuration does not modify the success of the operation.

OPERATIONS ON THE LACRIMAL APPARATUS.

To **probe the nasal duct,** a lacrimal probe, of which various sizes should be at command, is necessary. If possible, pass a probe of middle size through the punctum of the lower lid after it has been dilated, and push it horizontally inward in the canaliculus until the bony wall is felt.

When great difficulty is experienced in passing the probe through the canaliculus, it is necessary to **slit it up.** This can be accomplished by passing the point of a canaliculus knife vertically into the puncture, and after turning the instrument (with the cutting edge directed upward) in a line with the canaliculus, it is pushed through until the resistance of the lacrimal bone is felt. The handle of the knife is then depressed and slightly withdrawn, to divide the internal neck; and having been again introduced to the limit of the sac, the instrument is brought into a vertical position, the cutting edge of the knife being so held that the incision is slightly directed inward. This favors drainage. This operation is to be done only when absolutely necessary to permit the passage of the probe.

When the end of the probe touches the inner wall of the sac, it is turned vertically upward, and by firm and constant pressure pushed downward, slightly outward, and backward through the nasal duct to the inferior meatus of the nose. Syringing of the nasal duct is contra-indicated immediately after any cutting operation of these parts.

To **divide a stricture** of the nasal duct, a strong, narrow knife may be used like a probe, and pushed through the nasal duct, care being taken not to make a false passage. The operation for cutting a stricture of the nasal duct is only a preliminary one for the passage of the lacrimal probes, which should be used long enough to establish a permanent opening.

TENOTOMY AND ADVANCEMENT.

Tenotomy of the *internal rectus* is frequently performed; the *external rectus* is less seldom operated on; and the other muscles are rarely touched.

To do an ordinary tenotomy the following instruments are necessary: a speculum, fixing forceps, Stevens's straight forceps, large and small squint hooks, and curved strabismus scissors.

A horizontal fold of conjunctiva is grasped by the forceps over the insertion of the muscle, and with the scissors an incision is made at right angles to the fold. This cut is then enlarged transversely across the insertion of the tendon by a snip of the scissors above and below. The conjunctiva is then pushed aside, which brings the capsule of Tenon into view. This is to be incised. Holding the wound open with the small forceps, the hook with the end downward is passed through the wound and kept in contact with the sclerotic. When the bent part of the hook is in the wound the instrument is turned around so that the end of the hook is upward. The end of the hook being next to the sclerotic when the instrument is brought upward, the hook must pass between it and the tendon. The tendon being engaged, the forceps can be dispensed with, and after transferring the hook to the left hand the scissors is passed into the wound between the sclerotic and the hook and the fibers of the tendon cut. The hook should be moved around toward the cornea, above and below, to be certain that all fibers have been divided. If the conjunctival wound is large, one suture may be employed to close it; otherwise the wound may be left alone.

The effect of a tenotomy upon the *internal rectus* is likely to increase with time, and may amount to 4 mm.; the effect on the *external rectus*, on the other hand, diminishes with time and never exceeds 2 mm.

The effect of *graduated tenotomies*, in which the tendon is button-holed or only partially cut, is still uncertain, being highly prized by some and considered useless by others.

An *advancement* is done when the effect of a tenotomy is insufficient or excessive.

To perform an advancement, general anesthesia is often used but is not absolutely necessary unless the patient is unreliable and nervous and the instruments used for a tenotomy, with the addition of advancement forceps, will be required.

The tendon is brought into view by vertical incisions through the conjunctiva and the capsule of Tenon. After the tendon has been firmly fixed in the advancement forceps it may be cut near its insertion. A silk suture is carried through the middle of the cut end of the tendon from before backward (including the conjunctiva and Tenon's capsule), and the needle, passed beneath the conjunctiva and capsule of Tenon, is brought out near the lateral edge of the cornea. Similar sutures are passed through the upper and lower parts of the extremity of the tendon; the upper one, after being carried beneath the conjunctiva and fascia, is brought out above the upper margin of the cornea; the lower suture after a similar course is brought out at the lower margin of the cornea. The advancement forceps may now be removed. The middle suture is then tightened sufficiently to produce the required effect and tied. The upper and lower sutures are then tied and the operation is finished.

The eye should be bandaged for a few days and the sutures left in place, if possible, for five or six days.

An advancement of a muscle is usually combined with a tenotomy of the antagonist.

ENUCLEATION AND EVISCERATION.

Enucleation is the removal of the eyeball from the capsule of Tenon with preservation of the muscles, conjunctiva, and other orbital tissues. The *indications* for an enucleation are: injuries of such kind that the eye is inevitably lost, absolute glaucoma when other treatment is useless, malignant tumors, conditions from which sympathetic inflammation may occur, ectasis not amenable to treatment, ocular hemorrhage which cannot be arrested, and a blinded and disfiguring eye. The necessary *instruments* are: a speculum, strabismus scissors, heavy curved scissors, fixation forceps, and a large squint hook. General anesthesia is usually employed for this operation, but deep cocaine injections have been found satisfactory.

The ocular conjunctiva is divided around the cornea and as near as possible to it. The capsule of Tenon is then opened and the external, superior, and inferior recti muscles and connective tissue are divided close to the bulb, with or without the use of the squint hook. When the three recti have been divided, the eye can easily be dislocated forward, and with the heavy curved scissors the optic nerve can be cut. The internal rectus and the obliques are then cut close to the bulb, and the operation is finished. Hemorrhage is not considerable, and is controlled by pressure. The conjunctival wound may be closed by sutures, but this is not necessary. An artificial eye may be worn three weeks after the operation.

A simpler method of enucleation is this: After completing the circumcorneal incision through the conjunctiva and releasing it from the eyeball, divide the muscles as close as possible to their insertion. Then place the loop of a snare (Tyding's tonsil snare answers admirably) between the loosened conjunctiva and the eyeball and depressing the wire as much as possible sever the optic nerve and adherent parts. This will be almost bloodless.

An **evisceration** of the bulb consists in removal of everything within the sclera, particular care being taken that none of the choroid is left. The cornea and adjacent sclera is cut away and the contents of the bulb are removed with a spoon. The conjunctival edges are brought into apposition with sutures; but the stump is not better than that left after enucleation unless a glass ball be included in the scleral cavity. This, however, cannot be well tolerated and is often expelled.

Exenteration of the orbit is called for more particularly in malignant disease. General anesthesia is necessary. The lids must be separated by an incision at the outer commissure extending beyond the orbital wall. With a scalpel the soft parts behind the lids are incised to the edge of the orbit. The periosteum is then elevated from the entire orbital wall to the apex of the orbit. The contents of the orbit may then be removed and a snare will be found useful for this. Hemorrhage may be controlled by cautery.

CORNEAL OPERATIONS.

Paracentesis.—Paracentesis of the cornea is performed with a needle, which is thrust preferably into its lower and outer part. When the needle enters the anterior chamber, the instrument should be brought into a plane parallel with the iris, to avoid wounding it.

Corneal Section.—The corneal section made for relief of bad ulcers is performed with a Graefe knife. This is to be plunged through sound corneal tissue into the anterior chamber, across which it is carried and pushed out of the cornea in sound tissue at the opposite side. The wound heals quickly unless it is probed too often.

OPERATIONS ON IRIS AND SCLERA.

Iridectomy.—Iridectomy is an operation whereby a portion of the iris is removed.

The *indications* for such an operation are: to make an artificial pupil; to diminish the intra-ocular pressure; to prevent the recurrence of iritis; to remove foreign bodies; and to facilitate the extraction of a cataract.

An *artificial pupil* is desirable, the retina and nerve being sound, when there are dense opacities of the cornea or lens which do not extend to the periphery; when the pupil is occluded; or when other treatment for a dislocated lens is useless. *Increase of tension* due to primary or secondary glaucoma is often relieved by an iridectomy, which, if made for this purpose, necessitates the removal of a large portion of the iris as near as possible to its ciliary attachment. After subsidence of inflammation following a *recurring iritis* an iridectomy at times prevents a recurrence. *Small cysts* and *tumors* of the iris and *foreign bodies* often require the removal of the segment of iris in which they are developing or imbedded. A preliminary iridectomy may be performed to facilitate an *extraction of a cataract* when there are old adhesions and other complications. It may also be done to *hasten the opacity* of a cataract. After an iridectomy for this purpose, the lens should be kneaded by massaging the cornea with a cataract spatula.

The *instruments* necessary to perform an iridectomy are: a spring speculum, to keep the lids apart; fixation forceps, to steady the eyeball; a bent triangular keratome, to incise the cornea; iris forceps, to grasp the iris and withdraw it through the corneal wound; curved scissors for excising the part of the iris withdrawn; and a spatula to replace the iris.

The **site** of the iridectomy depends upon the reason for which it is to be made. When for optical purposes, the lower nasal side should be selected, the coloboma should be narrow, and the excised portion of the iris not too large (Fig. 27).

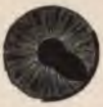

Fig. 27.—Iridectomy downward and inward for artificial pupil. (Nettleship.)

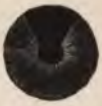

Fig. 28.—Iridectomy for glaucoma. (De Wecker.)

An iridectomy is generally made in the upper part of the iris, since the upper lid will partially cover the coloboma. An iridectomy for glaucoma should be broad and the iris removed near its ciliary attachment (Fig. 28). In order to do this the corneal incision must be as near as possible to the plane of the iris.

To perform an iridectomy, the operator stands behind the patient, and after the lids are separated by a speculum, the eyeball is steadied with fixation forceps at a point opposite the proposed incision, which is then made. The point of the keratome should first be pushed through the cornea perpendicularly, and as soon as it is seen in the anterior chamber the plane of the keratome must be directed from the lens in order that the capsule may not be injured. This should be near the plane of the iris when for relief of tension and in the limbus ordinarily, unless the operation be undertaken for optical reasons, when it may be even inside the limbus. The iris is grasped with the forceps, which has been introduced through the wound, and withdrawn. With the scissors this part is excised, and after replacing the cut edges of the iris, if necessary, the operation is finished. The eye should be kept bandaged for about a week.

Iridotomy.—Iridotomy is the operation for dividing the iris without removing a part of it. Its application is limited, and then it is not very successful. Most cases requiring it are eyes which are blinded from exudate after cataract extraction. It may be done with a Graefe knife, which is first pushed through the cornea, when the iris and opaque membrane may be so incised that the pupil becomes a slit.

Scleral Trephine.—The scleral trephine is performed under local anesthesia. An incision parallel with the upper border of the sclerocorneal margin about $\frac{5}{8}$ of an inch in length and $\frac{1}{4}$ of an inch from it is made through the conjunctiva. This is dissected well down to the cornea. With the trephine a small circular piece of the sclera at the limbus is cut through and removed. The iris usually presents in the wound and a small piece is excised which buttonholes it. The conjunctival flap is then replaced and secured by two or three sutures. This operation secures permanent drainage of the eye and is recommended in glaucoma. Its disadvantage is later infection due to destruction of the conjunctiva exposing the trephined area.

Anterior Sclerotomy.—Anterior sclerotomy is an operation for relieving the increase of intra-ocular tension. To perform this, a spring speculum, fixation forceps, and a Graefe knife are necessary. Before operating the pupil should be contracted by eserine, which is even indicated in glaucoma, in order that the iris may not prolapse. The eyeball being fixed, the Graefe knife, the sharp edge upward, is pushed through the sclera near the corneal margin as close as possible to the plane of the iris on the upper temporal side; and after crossing the anterior chamber should leave the eye at a corresponding place on the other side. After a few strokes of the knife it should be slowly withdrawn before a flap has been made. A bridge of tissue lies between the incisions, one being on each side. This operation, as a rule, is not as satisfactory as an iridectomy.

Posterior Sclerotomy.—Posterior sclerotomy may be done in glaucoma or in retinal detachment. A Graefe's knife is pushed through the sclera in the lower outer quadrant of the eye behind the ciliary body, between the external and inferior rectus muscles, and the incision should be anteroposterior and about $\frac{1}{3}$ of an inch in length and $\frac{1}{3}$ of an inch in depth.

CATARACT OPERATIONS

The *linear* and *flap extractions* are the operations for removal of cataracts.

Before operating for cataract it is absolutely necessary to determine whether the case is likely to be favorable. In a darkened room the perception and projection of light should be demonstrated, and if found to be good, the outlook is good. The pupillary reaction to light should also be good. Antecedent intra-ocular disease renders the prognosis unfavorable, and it goes without saying that if there is more than an opaque lens at the bottom of the trouble, an operation is of doubtful utility or even useless. A bacteriological examination of the conjunctival secretions should precede every operation. The blood-pressure should also be ascertained and the urine examined with reference to albumin and sugar. A very high blood-pressure or a diabetic condition being frequently unfavorable, the proper treatment to relieve these conditions should be instituted.

Linear Extraction.—The linear extraction is applicable only for soft or membranous cataracts. Before performing this operation the pupil should be dilated with atropine. For a soft cataract a small opening about $\frac{1}{3}$ of an inch wide is made with a keratome within the margin of the cornea at its lower border. The capsule is then opened, if this has not already been done by a previous operation, and all pieces of lens substance are forced out of the eye by pressing on the lower part of it. To remove cataractous membranes after making the corneal opening a hook, punch or forceps may be tried.

For removing hard cataracts the **flap extraction** is necessary: The *first step* of the operation is the incision, which should be large enough to give exit to the hardened lens. This is best done with Graefe's straight knife of which the cutting edge, directed upward, is pushed through the limbus at the upper temporal side, and after passing through the anterior chamber should come out of the eye at a corresponding point on the nasal side. This should include $\frac{2}{5}$ to $\frac{1}{2}$ of the sclerocorneal margin. By making a few strokes with the knife the incision is completed, and it is well to include a bit of conjunctiva in the flap.

The *second step* is an iridectomy, and the *third* is the opening of the anterior capsule, which is done with a cystotome; and the *last step* is the expulsion of the lens.

After this any extraneous material in the anterior chamber is to be kneaded out by stroking the eye with a spatula, the iris is to be neatly replaced if necessary, the conjunctival wound nicely coapted, and the eyes bandaged.

The extraction may also be performed without an iridectomy (simple extraction), but the beginner will do better by employing the other (combined) method.

The *instruments* necessary to perform an extraction of a hard cataract are the same as required for an iridectomy (except that a Graefe knife is used instead of a keratome); a cystotome, to tear capsule; two scoops; and iris repositor.

The Indian operation, or intracapsular, practised many thousand times by Col. Smith, of Amritsar, India, removes the lens in its capsule and may be done at any time before maturity as well as later. If successfully performed, there is no capsule or cortical matter left in the eye, and complications less often occur which require treatment.

The lids must be held from the eyeball by lid hooks (Figs. 29 and 30) in the hands of a trained assistant, and at no time is there to be any pressure on the eyeball. The incision is made to include the upper half of the cornea with one sweep of the knife after transfixion. An iridectomy is made as usual and then the lens is to be freed from the suspensory ligament by pressure on the ciliary region below, the lens either presenting with the upper part first or tumbling with the inferior part first. If there is threatened or actual loss of vitreous, the lens may be removed by a needle when it presents. The use of the lid hooks will be found a valuable substitute for the speculum, whatever may be the technic. The loss of vitreous will be eliminated by the proper use of these, and if the lens has been removed in its capsule, a subsequent needling will not be necessary. *After extraction* the closed lids of both eyes should be covered with yellow oxide salve and bandaged and the patient kept in bed for a week or nine days. If all goes well, no special pain nor discomfort, the bandage may be left alone for nine days. In some cases the non-operated eye may be left open after the sixth or seventh day, and if the bandage proves very uncomfortable, the secretions annoying or itching intolerable, the bandage may be removed daily, the external parts very carefully washed and annointed as necessary. After the ninth day the patient should wear a shade during the day and a bandage at night and on the fourteenth day he may usually leave the hospital. Glasses may be fitted about six weeks after the operation.

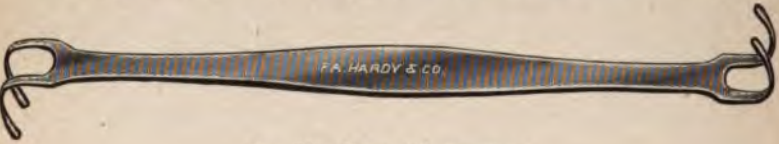

Fig. 29.—Upper lid retractor.

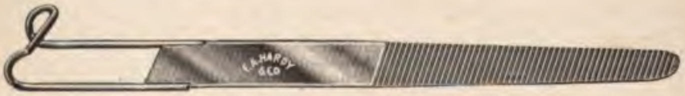

Fig. 30.—Lower lid retractor.

Soft cataracts may also be removed by *solution* or by *suction*.

Discission.—The operation of discission causes absorption of the lens substance by exposing it to the action of the aqueous. To perform this operation the pupil must be dilated, general or local anesthesia (according as the patient is unruly or not) employed, and after separation of the lids a cataract needle is thrust into the cornea near the lower and temporal border (Fig. 31). The point of the instrument is pushed through the capsule into the lens substance, and after this has been gently disturbed, the needle is withdrawn. The danger of the operation lies in the possibility of wounding the iris, and this is to be kept in mind. The *after-treatment* consists in rest, use of atropine, and, if there be considerable reaction, application of iced water and leeches.

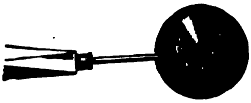

FIG. 31.—Discission of cataract. (Nettleship.)

For removal by **suction,** a cataract must be liquid. A discission is first performed and the nose of the syringe is passed through an opening in the cornea made with a keratome. When the end of the instrument is within the capsule, the contents can be removed by suction. The *after-treatment* is the same as for discission.

THE EAR.

ANATOMY OF THE EAR.

Anatomically the ear is divided into three parts, viz:

(*a*) The external ear.
(*b*) The middle ear.
(*c*) The internal ear.

Physiologically the ear may be divided into two parts, namely:

(*a*) The conduction apparatus, by means of which sound waves are carried to the terminal filaments of the auditory nerve.

(*b*) The perception apparatus, by means of which sound waves are received and the impression conveyed to the brain centres, where they are recognized as sound.

The conduction apparatus is composed of the external and middle ear and the perception apparatus consists of the internal ear.

The External Ear.—The external ear includes the pinna (Fig. 32) and the external auditory canal.

The external auditory canal is divided into two parts, an external or cartilaginous portion, and an inner, osseous portion. Dense fibrous tissue connects the two sections.

The length of the canal averages from 35 to 38 mm. It runs forward and inward in an oblique direction and presents a curve which is higher near the middle of its length than at either extremity. Throughout its entire extent it is lined by cutis closely adherent to the underlying tissue.

The sebaceous glands are limited to the cartilaginous portion of the canal, hence furunculosis is confined to this area.

The Middle Ear.—The middle ear embraces:

1. Tympanic membrane, or drum head.
2. The tympanic cavity (Fig. 34) and its contents (the ossicles, ligaments, muscles, etc.).
3. Eustachian tube.
4. Antrum.
5. Mastoid cells.

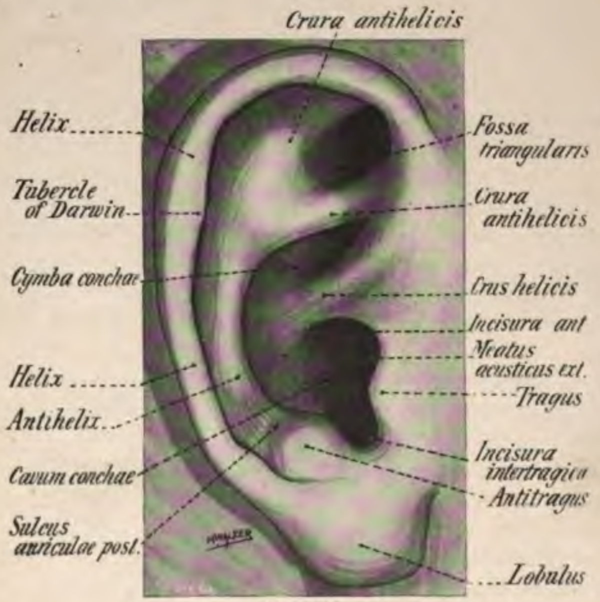

FIG. 32

The Tympanic Membrane.—The tympanic membrane (Fig. 33) forms the outer wall of the middle ear. The size of the drum membrane is about 8 mm. wide, 9 mm. high and about $\frac{1}{10}$ mm. thick. It is not a plane surface but indrawn like a shallow funnel, the umbo forming the apex. It is inclined in both vertical and horizontal planes at about 45 degrees so that the superior and posterior quadrants are nearer the observer than the anterior and inferior quadrants. In infants this inclination is more marked, so that the membrane more nearly approaches the horizontal. The tympanic membrane is inserted in a small groove called the annulus tympanicus except at the upper part of the membrane where it is attached to the squama in the incisura Rivini. The membrana tympani is composed of three layers: the outer one being a reflection of the skin of the meatus made up of layers of flat epithelium with cylindric cells in the deepest layers. The inner layer is a reflection of the mucous membrane of the middle ear consisting of simple cuboidal epithelium, and the middle or fibrous layer consists of an outer radiating and an inner circular layer of fibrous tissue poor in elastic fibers. In that portion of the drum membrane known as the **pars flaccida** or Shrapnell's membrane, a small concentric-shaped field situated between the processus brevis and the incisura Rivini, the middle or fibrous layer is absent. The outer skin layer and the inner mucous membrane layer form the pars flaccida. The remaining portion of the drum membrane or the **pars tensa** is made up of the three layers as given above.

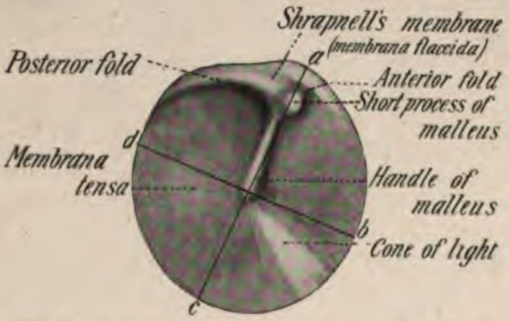

FIG. 33.—The drum membrane. *a, b,* anterior superior quadrant; *b, c,* anterior inferior quadrant; *c, d,* posterior inferior quadrant; *d, a,* posterior superior quadrant.

The tympanic membrane presents certain landmarks by which its presence is recognized. A prominent one is the **short process** of the malleus which is seen as a more or less prominent grayish-white point in the upper and anterior portion. Extending downward and backward from the short process to the middle of the drum membrane is the handle of the malleus or the **manubrium**. At the tip of the manubrium is a flattened area called the **umbo**. Upon looking at the drum membrane by means of reflected light a cone-shaped **light reflex** can be seen, having its apex at the umbo and extending downward and forward. From the short process of the malleus extend two folds in the membrane. One passing forward is called the **anterior fold**, and the other, much longer, is called the **posterior fold**. These folds form the lower boundary of Shrapnell's membrane.

In addition to the landmarks just mentioned one is sometimes able to see through the membrane some of the contents of the tympanum. Of these the most prominent is the long process of the incus. This is seen projecting downward to a point about the centre of the posterior superior quadrant. From the tip of the long process of the incus extending inward the shadow of the incus can sometimes be seen. At times the chorda tympani nerve is observed, passing from behind forward across the tympanum just below the level of the posterior fold.

For convenience of describing, the drum membrane is divided into quadrants. A line drawn through the short process and the umbo, and another line at right angles to this and passing through the umbo, will divide the drum membrane into four parts, an anterior superior, and anterior inferior, a posterior superior, and a posterior inferior quadrant (Fig. 33). Behind the anterior superior quadrant lies the opening of the Eustachian tube, the anterior mucous pouch of the drum and the canal for the tensor tympani. Behind the anterior inferior quadrant lies the carotid canal. Behind the posterior superior quadrant is situated the stapes, the chorda tympani nerve, the long process of the incus, the posterior mucous pocket of the drum membrane, and the pyramid containing the stapedius muscle.

The Tympanic Cavity.—The tympanic cavity (Fig. 34) may be described as a flattened cylinder. It is divided into three parts, namely:

1. The epitympanic space (vault, attic), or that part lying above the level of the upper margin of the membrana tensa.

2. The atrium, or that portion of the tympanic cavity situated between the levels of the upper and lower margin of the membrana tensa.

3. The hypotympanic space, or that portion of the middle ear cavity lying below the inferior margin of the drum membrane.

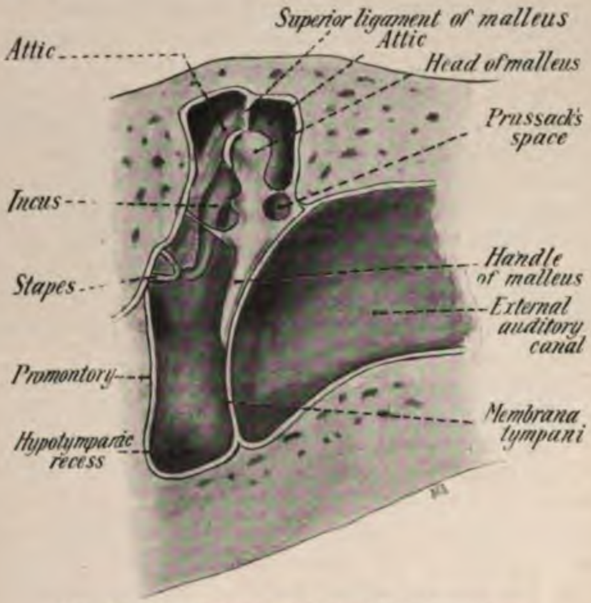

FIG. 34.—Coronal section through the tympanum.

For ease of description six walls are given, namely: an outer (lateral) wall which separates the tympanic cavity from the external auditory canal. This wall is formed by the membrana tympani and its bony framework: an inner (mesial) wall which is the outer wall of the internal ear and is formed by the outer surface of the petrous bone through which the middle and internal ears are joined by the oval and round windows. The promontory, a somewhat rounded eminence of hard bone formed by the first turn of the cochlea is located near the middle of the mesial wall. Anteriorly, the promontory merges into the wall of the Eustachian tube. Posteriorly and below, the fenestra rotunda is situated. Posteriorly and above, the fenestra ovalis can be seen which in the living subject is closed by the foot plate of the stapes. The outer wall of the Fallopian canal, which lodges the facial nerve, forms a well-marked ridge (facial ridge) which runs from before backward just above the middle of the mesial wall. Just above the Eustachian tube is the canal for the tensor tympani muscle. Above the posterior end of the facial ridge is a small linear bulging of smooth dense bone which denotes the position of the horizontal (external) semicircular canal.

The upper wall (roof) of the tympanic cavity is formed by the tegmen tympani (paries tegmentalis) which separates the middle-ear cavity from the middle cranial cavity. This plate of bone is frequently very thin and quite often dehiscences in its structure are found, in which event the dura is in contact with the lining mucous membrane of the middle-ear cavity.

The inferior wall (floor) separates the tympanic cavity from the jugular bulb and from the carotid artery. The bone forming the floor is of a diploic nature; however, it is sometimes very thin. Dehiscences in this bone have been observed.

The anterior wall contains the opening of the Eustachian tube, and the canal for the tensor tympani muscle. Below the inferior edge of the Eustachian orifice the anterior tympanic wall is in relation to the canal for the internal carotid artery.

The posterior wall of the tympanic cavity presents in its upper portion the opening of the tympanic cavity into the mastoid antrum known as the **aditus ad antrum**. It is a large, somewhat triangular-shaped opening with the apex directed downward. Beneath the aditus ad antrum on the posterior wall of the atrium is situated the pyramidal process within which the stapedial muscle is located.

The Ossicles.—The ossicles (Fig. 35) are three in number the malleus, incus, and stapes. They form a chain connecting the membrana tympani with the oval window of the labyrinth. The *malleus,* the largest of the three, presents a head which lies in the vault behind the membrana flaccida and a handle (manubrium mallei) which is attached to the inner surface of the drum membrane. Connecting the head and the handle of the malleus is a constricted portion called the neck. Anteriorly the neck presents a long process (processus gracilis). The short process extends outwardly from the base of the handle.

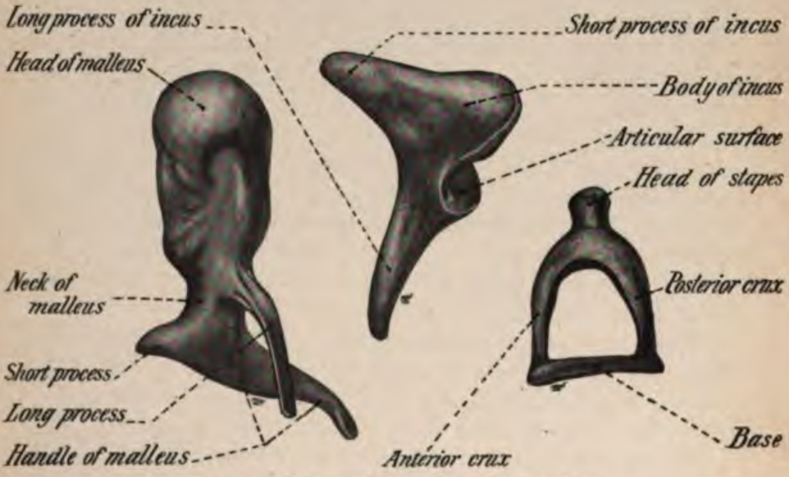

Fig. 35.—The ossicles. (After Spalteholz.)

The *incus* presents a body and two processes. The body of the incus occupies the posterior portion of the vault. The anterior surface of the body of the incus articulates with the head of the malleus. The short process of the incus extends backward from the body and articulates with a small facet on the posterior wall of the vault just below the aditus ad antrum. The long process of the incus extends downward and backward and articulates with the stapes. This process is nearly parallel with the handle of the malleus.

The *stapes* or *stirrup* presents a foot plate which is attached to the oval window by an annular ligament, two cruræ or arms arise and unite, forming a neck. Above the neck is a small enlargement called the head.

Five ligaments support the ossicles in the tympanic cavity, namely: anterior, superior and lateral ligaments of the malleus, the superior ligament of the incus and the ligament of the short processes of the incus. In addition the foot plate of the stapes is attached to the oval window by the annular ligament. A loose capsular ligament binds together the incus and malleus and another capsular ligament encloses the incudostapedial articulation. Folds of mucous membrane cover the ossicles and ligaments forming pouches or pockets. Three of these pouches, the anterior pouch, the posterior pouch and Prussak's space are in contact with the drum membrane.

Two muscles are located within the tympanic cavity, namely: the stapedius muscle which arises from the pyramidal bony excrescence on the posterior and internal tympanic walls and is inserted in the neck of the stapes, and the tensor tympani muscle which arises from just above the osseous portion of the Eustachian canal and from the upper wall of the cartilaginous portion of the Eustachian tube. The tendon of this muscle after passing backward and upward across the inner wall of the tympanum within a special bony canal emerges in front of the oval window. It then curves around the processus cochleariformis, then crosses to the lateral wall to be inserted into the inner and anterior surfaces of the handle of the malleus.

The **chorda tympani** nerve passes through the upper portion of the atrium between the handle of the malleus and the long process of the incus.

The Eustachian Tube.—The Eustachian tube connects the tympanic cavity of the middle ear with the nasopharynx. It is through this channel that the normal air pressure is maintained in the middle ear. It also is an important factor in the sound-conduction apparatus. It is about 36 mm. long, extending from the tympanic cavity in a downward, forward and inward direction.

The tube is composed of an upper osseous third, or that third nearest to the tympanic cavity, and a lower membrano-cartilaginous two-thirds, or that portion nearest the nasopharynx. The tube is somewhat expanded at either end and is constricted at the junction of the osseous and cartilaginous portions to form the isthmus. The osseous portion is in relation above with the canal for the tensor tympani muscle and internally with the canal for the internal carotid artery. The membranocartilaginous portion is made up of an inner membranous part and an outer cartilaginous wall. Normally the lower portion of the tube is closed except when the palatal muscles act upon it as in swallowing. Three muscles act upon the tube: (1) the **levator palati** which arises from the under side of the tip of the petrous bone and from the lower and outer surface of the Eustachian cartilage and is inserted into the soft palate; (2) the **tensor palati** which arises from the scaphoid fossa, from the spine of the sphenoid, and from the anterior portion of the inner membranous wall of the tube. This muscle is attached to the anterior surface of the soft palate; (3) the **salpingopharyngeus** which arises from the cartilaginous portion of the tube is inserted into the post-pharyngeal wall. The levator palati is supplied by the great superficial petrosal nerve, a branch of the facial. The tensor palati is supplied by the inferior maxillary division of the fifth nerve.

The Antrum.—The antrum is a small cavity or air cell the size of a pea lying between the mastoid and the tympanic cavity. It is the first mastoid cell and is present at birth. It communicates with the middle-ear cavity by way of the **aditus ad antrum.** The roof of the antrum is a continuation of the roof of the tympanic cavity. The mastoid cells drain into the antrum. The ciliated epithelium lining the mastoid cells, antrum, tympanum, and the Eustachian tube propels the secretions successively through these parts to the nasopharynx.

The Mastoid Process.—The mastoid process consists of an outer shell or cortex of hard bone and an inner mass of communicating air cells or of diploic bone. Accordingly three types may be observed.

1. The pneumatic mastoid may consist of large or small cells which communicate with each other by means of apertures. A large bulbous mastoid is nearly always of this type.

2. The diploic mastoid which is always present in infants may persist in adults. This type is richly supplied with blood and lymph spaces.

3. The mixed type is a combination of the two above.

The Internal Ear.—The internal ear or perceptive apparatus consists of (1) the osseous labyrinth or the outer bony capsule; (2) the membranous labyrinth, with its nerve-end organs; and (3) the auditory nerve and its ganglia.

The Osseous Labyrinth.—The osseous labyrinth may be divided into (*a*) the vestibule, (*b*) the semicircular canals, (*c*) the cochlea.

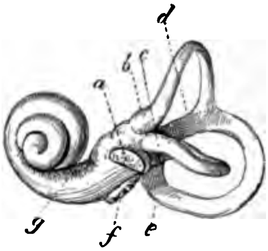

FIG. 36.—External view of a cast of the left labyrinth: *f*, fenestra cochleæ, or round window; *a*, fenestra vestibuli, or oval window; *b*, ampulla of superior semicircular canal; *c*, ampulla of posterior semicircular canal; *d*, common shaft of union of these two canals; *c*, ampulla of the horizontal semicircular canal; *g*, tractus spiralis foraminosus. (Henle.)

The Vestibule.—The vestibule is an irregularly shaped space about 6 mm. in the anterior posterior direction, about 4 mm. in width and approximately 4 mm. in height. We may assume the vestibule to have six walls. Communication between the tympanic cavity and the vestibule is maintained through the vestibular window which in life is closed by the foot plate of the stapes. The vestibular window is in the lateral wall of the vestibule. Running over the roof and anterior wall to the floor is a slight ridge called the crista vestibulæ. Behind the crista vestibulæ is an elliptical depression called the recessus ellipticus, which contains the utriculus. In front of the ridge is a small spherical depression called the recessus sphericus. The recessus sphericus receives the sacculi in the living subject. The upper and inner walls of the vestibulum have no openings. The anterior wall lodges the area cribrosa sacculi which is situated in the centre of the recessus sphericus. The posterior wall contains the opening of the small end of the horizontal semicircular canal and the terminal aperture of the lateral ampulla. At the juncture of the roof and the posterior wall the terminal ostia of the upper ampulla and the common crus of the superior and posterior semicircular canals are located. At the junction of the floor and the anterior and outer walls is the opening into the scala vestibuli. The roof contains the ampullæ of the horizontal and of the anterior vertical canals. The ampulla of the posterior vertical canal is found in the floor.

The Semicircular Canals.—The semicircular canals are three in number: the horizontal (external), the anterior vertical and the posterior vertical. The plane of each canal lies at right angles to the planes of the other two canals. With the head erect and chin in, the external canal approaches the horizontal plane very closely. The anterior vertical canal lies in a vertical plane whose direction directed from within runs outward and forward. The plane of the posterior vertical canal is directed outward and backward. Each canal is about 1 mm. in diameter except at its end where it expands into a bulbous enlargement called the ampulla, which is about 2 mm. in diameter.

The Cochlea.—The cochlea is a bony spiral formed by two and one-half convolutions which terminate in a flat cupola. The length of the tube is about one and one-half inches. The convolutions are differentiated as basal, medial, and apical. The axial part of the cochlea known as the modiolus extends from the base to the apex. Passing outward from the modiolus is a spiral lamina (lamina spiralis) which divides every convolution into an upper or outer channel (scala vestibuli) and a lower or inner channel (scala tympani).

The Membranous Labyrinth.—Enclosed within the osseous labyrinth and differing somewhat in shape is the membranous labyrinth. Instead of the single cavity (vestibule) of the former, the membranous labyrinth presents two sacs —the utriculus and the sacculus—united by a narrow duct, the utriculosacular duct. The membranous labyrinth is surrounded by a supporting and a protecting fluid, the perilymph. Enclosed within the labyrinth is a similar fluid called the endolymph.

The membranous vestibule consists of the saccule and the utricle. The saccule communicates with the scala media of the cochlea and indirectly with the utricle. The utricle communicates with the three semicircular canals by five openings. A small tube or duct is given off the utricle. This duct unites with a similar one from the saccule to form the aqueductus vestibuli. The aqueductus vestibuli traverses the inner wall of the bony vestibule and emerges upon the posterior surface of the petrous bone where the duct expands to form the saccus endolymphaticus.

The *membranous semicircular canals* are attached at their convex surfaces to the periosteum of the bony canals, which they only partly fill. The perilymphatic space around the membranous canals is traversed by connective-tissue trabeculæ.

The membranous portion of the cochlea consists of the cochlear duct and two perilymphatic canals. The cochlear duct is a long tube closed at both ends, one end forming the cecum vestibulare, and the other the cupolar extremity or the cecum cupolare. The cochlear duct communicates with the sacculus by means of the canalis reuniens. The cochlear duct lies above the lamina spiralis, and runs between and parallel with the two perilymphatic canals of the cochlea, that is, with the scala vestibuli and scala tympani.

The *nerve end organs* of the membranous labyrinth are distributed over certain spots known as the maculæ, cristæ, and papilla spiralis. One of these areas located within the recess of the utriculus is known as the macula acustica utriculi; and another within the sacculus is called the macula acustica sacculi. The ampullæ of the semicircular canals have these nerve end organs and in this location are known as cristæ ampullares sup., cristæ ampullares lat., and cristæ ampullares inf. The terminal arborization of the acoustic nerve in the cochlea is known as the papilla spiralis cochlear, or the organ of Corti.

EXAMINATION OF THE EAR.

The examination of the ear is made under three heads: (1) History. (2) Objective examination. (3) Functional examination.

History.—The first essential in the examination of a patient for ear trouble is to obtain a brief but complete history of the condition for which the patient seeks relief. After obtaining the patient's name, address, age, occupation, nationality, and conditions of life, the chief complaint of the patient should be recorded, preferably in the patient's own words. Inquire especially concerning pain, deafness, discharge, and vertigo.

The family history may now be obtained with special emphasis placed upon the occurrence of deafness in other members of the family and upon the possibility of hereditary transmission of specific diseases.

The personal history should reveal the frequency of colds, tonsillitis, whether the patient has any constitutional disease such as syphilis or tuberculosis, the occurrence of the acute infectious diseases either in childhood or in later life. Previous attacks of earache and discharge should be recorded with their frequency and duration.

The history of the present trouble is of much importance and should be gone into in more detail.

1. The onset, whether abrupt or insidious.
2. The supposed cause.
3. The course, whether continuous or intermittent, getting better or worse.
4. Associated symptoms such as pain, deafness, discharge, tinnitus, vertigo, fever, headache, etc.

Objective Examination.—Inspection of the patient reveals much concerning his physical condition. The breathing should be noted, whether a mouth-breather or not. Malformations of the auricle, redness and edema of the postauricular region, or displacement of the auricle due to inflammatory reaction should be looked for. Outward displacement of the auricle in children, when associated with signs of inflammation, point very strongly to a mastoid involvement.

In adults, however, the probabilities are that the displacement and edema are due to a furunculosis of the external canal, especially if movement of the auricle causes pain.

Palpation of the mastoid frequently reveals the presence of edema, fluctuating areas, or tenderness.

Examination by Reflected Light.—For the objective examination of the ear certain instruments are required.

1. *Chair.*—This should have a high back and a comfortable head rest. A chair with a reclining back is very desirable at times. The observer uses a stool with a revolving top.

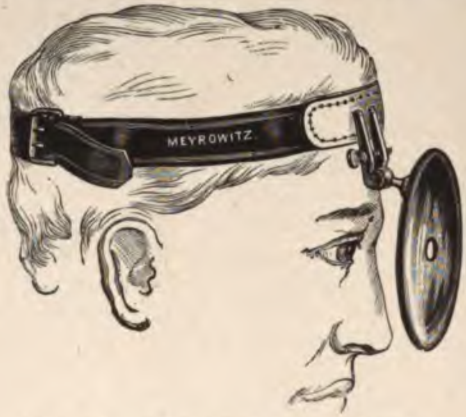

Fig. 37.—Indirect illumination of the ear.

2. *Light.*—This may be either gas or electricity. If gas is utilized, an opaque, unglazed chimney with an opening one inch wide and two or three inches long should be used. The electric bulb should be of ground glass with a clear circular area about two inches in diameter. The candle power should be sufficient to insure a strong light.

3. The *head mirror* should be from 3 to 3½ inches in diameter and should have a focal distance of 8 to 12 inches. For the nose and throat the mirror may have a focal distance of 12 to 18 inches. The head band may be made of cloth, leather, fiber or metal. For office work the fiber has proved very satisfactory.

4. Three or four sizes of *ear speculums* should be obtained. The Gruber speculum has given satisfaction.

5. The *probe* should have a blunt tip and be made of soft metal so as to bend easily without breaking.

6. The *cotton carrier* or *applicator* should be slender and have a screw tip to retain the cotton firmly.

7. The *ear forceps* should be bayonet-shaped or bent at an angle of 45 degrees so that the hand will not obstruct the view.

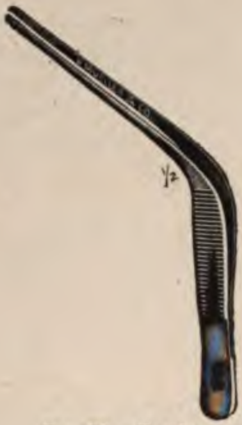

FIG. 38.—Ear forceps.

In examining the ear the patient is seated at right angles to the observer. The light should be about 10 inches from, and on a level with, the patient's ear. If the focal distance of the mirror is more than 12 inches, it is advisable to place the light somewhat to the rear of the patient's head. The student should cultivate the habit of sitting at the side, and not "stradling" or sitting with knees on each side of the patient. When inserting the ear speculum in the right ear for instance, the pinna is grasped with the thumb and forefinger of the left hand and gentle traction is exerted upward, backward and outward to straighten the canal. Before inserting the ear speculum the external meatus should be inspected for any traumatism, abrasion, discoloration, swelling, furuncle, etc. Having noted the condition of the external portion of the canal, the speculum, which should be approximately the size of the canal, is gently and slowly inserted with the right hand. If the hairs of the cartilaginous portion of the canal obstruct the view, it will be necessary to cover them with vaseline.

If pus is found in the canal, its source is generally from a suppurating middle ear. The perforation of the drum membrane in acute cases is frequently small and may require careful observation to detect it. If the pus pulsates it probably means an acute suppurative otitis media. A portion of the pus should be taken for a bacteriological examination.

The drum-head should be inspected, and the following conditions noted: The color should be a pinkish, pearly gray. If it is of a whitish, glistening color, with here and there thickened areas, the presence of sclerosis should be suspected. If it is of a dull, sodden, lustreless, pinkish color, otitis catarrhalis is probably present. If it is of a reddish-blue color with a velvety surface, it is indicative of acute otitis media; while if it is of a beefy-red color, with greenish or yellowish areas in the lower half, an acute suppurative otitis media is in active progress.

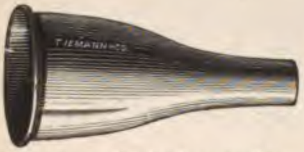

FIG. 39.—Gruber's speculum.

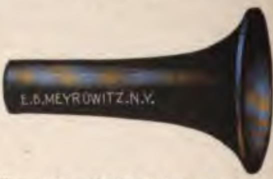
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FIG. 40.—Politzer's speculum.

It should be noted whether the membrane has the normal or an exaggerated concave surface, or is bulging outward. When the concavity of the drum-head is exaggerated, the handle of the malleus is rotated inward and backward, thereby making the short process stand out quite prominently as a whitish, glistening spot in the upper field of the drum-head. The cone of light which projects anteriorly from the umbo or lower end of the handle of the malleus, is broken or altogether wanting when extreme retraction is present.

Along the line of the handle of the malleus the bloodvessels may be quite prominent, and it should be determined whether this prominence is abnormal or due to the presence of the ear speculum within the meatus. After the speculum has been in the meatus for a few minutes, the manubrium, which at first had the normal appearance, becomes deeply congested; hence it is important that it should be examined immediately after inserting the speculum.

If the drum-head is *perforated*, the size, location, and character of the edges should be noted; the presence or absence of granulations, polypi, and necrotic bone should be determined.

The probe introduced into the middle-ear cavity, especially if it is curved and introduced upward into the *attic*, may be the means of demonstrating the presence of a necrotic or **foul odor**, when it would otherwise not be suspected. The probe thus affords another valuable means of diagnosis, in addition to the grating sensation produced by contact with bare bone.

Alcohol or *silver nitrate* may be used to demonstrate the presence or absence of perforation, by putting a few drops into the external meatus and applying air-pressure through a suitable meatus tip and forcing the medicament into the middle ear, and out through the Eustachian tube into the throat, where it is detected by the patient.

FUNCTIONAL TESTS OF HEARING.

The Voice.—This is one of the oldest methods of testing the hearing.

The Whispered Voice.—The whispered voice to be uniform should be the residual air whisper, or the whisper spoken after forced expiration. This, if given without force, is heard by a normal ear at a distance of about 21 feet. The forced whisper may be heard much farther, probably 60 to 75 feet. High-pitched words such as six, sees, tease, etc., are more readily heard than low-pitched ones such as, our, more, hood, etc.

Method.—The patient should be seated at one end of the room with the ear to be tested turned toward the physician. The patient should be requested to close his eyes and repeat the words or sentences spoken to him; or if the watch or acoumeter is used, to indicate by raising the hand when he distinctly hears it. The surgeon should approach from the normal hearing distance, using the voice as he approaches step by step. When he has determined at what distance the patient can hear the whispered voice, he should record it in feet or inches.

The Conversational and Loud Voice.—The conversational and loud voice may be used in the same way. In extremely deaf persons it is sometimes of advantage to test the hearing through a *speaking-tube* or other aid to hearing. A careful record should be kept of all these tests, so that they may be used for comparison at subsequent times.

The Watch.—The same general rules apply to this test.

Two watches may be used, one a high-pitched and the other a low-pitched ticker. They should be tested upon twenty or more normal ears, so as to establish the normal distance at which they should be heard. The ordinary watch is heard at a distance of from forty to sixty inches.

Having determined the normal distance, it should be taken as the denominator, the numerator being the actual distance at which it is heard. If, for instance, the denominator of the watch is fifty inches and the distance at which it is heard by the patient is ten inches, the record should be Watch = $\frac{10}{50}$.

If the watch is heard only upon *contact against the ear,* it should be recorded Watch = $\frac{c}{50}$; while if it is not heard by either air or contact, it should be recorded Watch = $\frac{0}{50}$.

Having a high- and low-sounding watch is of some value in differentiating middle-ear and labyrinthine disease, the high ticker being heard best in middle-ear disease, and the low ticker in labyrinthine.

The Acoumeter.—This is an instrument devised by Politzer for the purpose of giving to the specialist a ticker tuned to a certain pitch and intensity, whereby it is possible for the records of all observers to be uniform and easily compared.

This instrument (Fig. 41) can normally be heard in a quiet room at a distance of about forty feet. This should be taken as the denominator and the actual distance at which it is heard as the numerator.

Thus if it is heard at *eight feet,* it should be recorded P. Ac. $\frac{8}{40}$.

There is an attachment by means of which the instrument can be placed in contact with the mastoid process and the bone conduction thus tested.

The Galton Whistle.—This is an instrument, the cylinder of which can be lengthened or shortened by a simple contrivance and the pitch of the tone varied accordingly.

Its range is from the highest pitch that can be distinguished by the human ear (about 22,000 double vibrations per second) down to near the medium range.

Its purpose is to test the *upper limit* of hearing. It is of very great value in the diagnosis of labyrinthine disease, or disease of the auditory nerve where the upper limit of the hearing is considerably lowered.

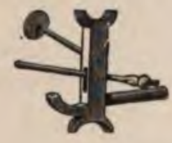

Fig. 41.—Politzer's acoumeter.

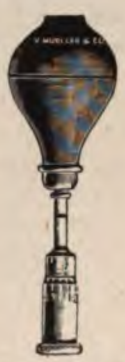

Fig. 42.—Galton whistle.

Konig's Rods.—Konig's rods may be used for the same purpose, although they are neither so convenient nor inexpensive as the Galton whistle.

The Monochord.—This instrument consists of a metal framework supporting a piano wire. A block is in contact with the wire. The position of the block regulates the pitch. The range of the Struycken-Schaefer monochord is from g′ to above the high limit.

The Barany Noise Apparatus.—The Barany noise apparatus is a clock-work buzzer designed to exclude all other sound in the ear on which the apparatus operates while the other ear is tested. This instrument enables the examiner to detect total or partial deafness in either ear.

Range of Hearing.—The normal range of hearing of musical tones in man is from 16 double vibrations per second, the low limit, to 22,097 double vibrations per second, the high limit. The range of hearing is determined by ascertaining the lowest and the highest musical tone that can be heard by the affected ear.

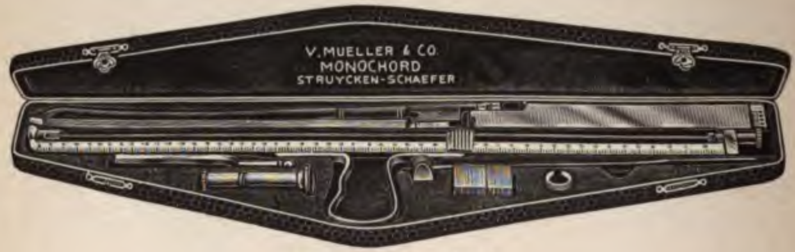

FIG. 43.—Monochard.

The best set of forks and whistles is undoubtedly the Bezold-Edelmann, as it is accurately constructed upon scientific principles. Every otologist should possess this set as it is the best one that will accurately test the patient's hearing throughout the entire range of normal tone perception. With this set one may determine the "islands of hearing" in deaf-mutes and if found these islands or remnants of hearing may be utilized to teach speech.

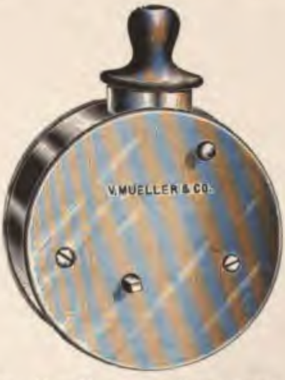

FIG. 44.—Barany noise apparatus.

The Hartman set of tuning-forks is frequently used to determine roughly the lower-tone limit, but they are of little use except in advanced catarrhal deafness.

In testing an ear for the low limit (16 d. v.) the examiner begins with the lowest fork, and if not heard, the next higher one is tried, until a fork is used that the patient hears. This is recorded as the low limit. The opposite ear is tested at the same time. The patient should have the eyes closed during the process.

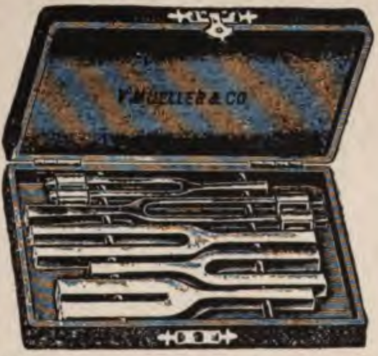

Fig. 45.—Hartman's set of tuning-forks.

The high limit (22,097 d. v.) may be determined most satisfactorily by means of the Galton whistle (Fig. 42). Begin above the limit and reduce until the note is perceived. The opposite ear should be firmly closed. The monochord (Fig. 43) may be used for testing the high limit, although not so convenient as the whistle. The bone conduction for high tones can be found with this instrument, a distinct advantage over the whistle.

The Bezold Tone Scale in Evenly Regulated Temperature (According to Edelmann).

C_2 — 16.165
G^2 — 24.221
D^1 — 36.290
A^1 — 54.375
F♯ — 91.447
A —108.750 (used for Schwabach test)
c^4 —2069.220
d♯ — 153.796
a — 217.500
d^1♯ — 307.593
a^1 — 435.000
d^2♯ — 615.185

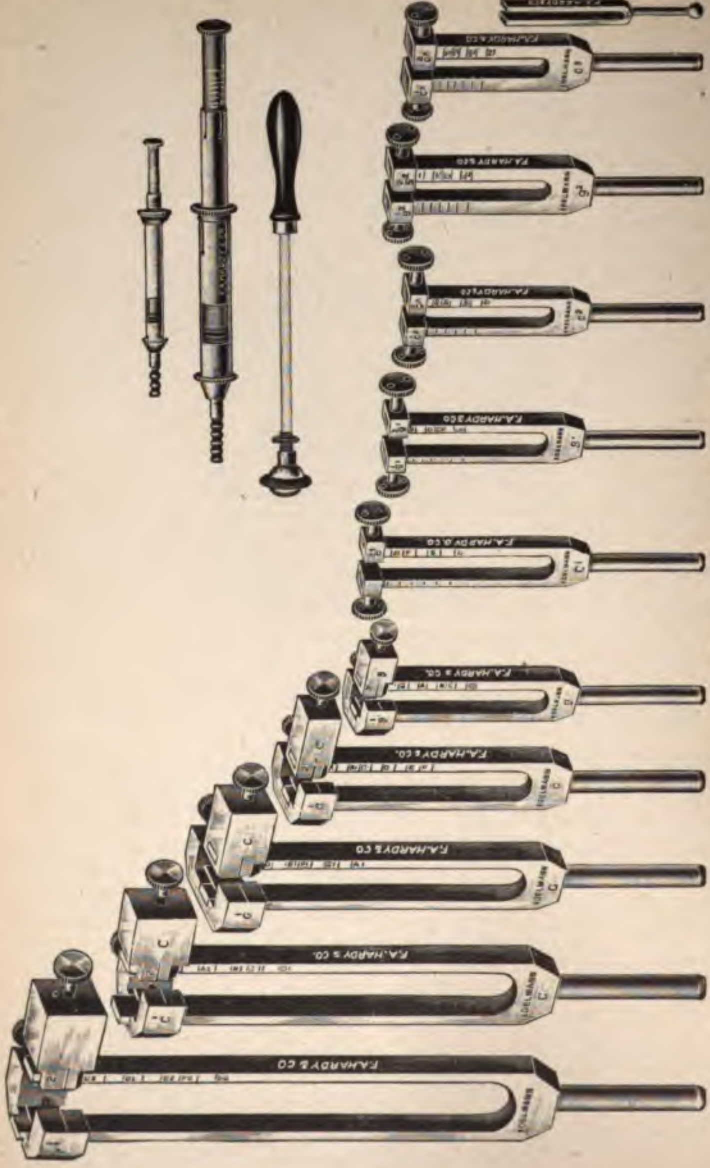

Fig. 46.—Bezold-Edelmann's set of tuning-forks.

The significance of testing the range of hearing is in the fact that in disease of the conduction apparatus the power to perceive low tones is diminished and in disease of the perception apparatus the ability to hear high tones is lessened.

In cases of extreme middle-ear deafness the patient may not hear lower tones than 2048 d. v., while he may distinctly hear the notes of the Galton whistle. In labyrinthine disease the upper notes of the Galton whistle are lost, while the low-sounding tuning-forks may be heard.

The Weber Test.—This test is made for the purpose of determining the *relative hearing of the two ears by bone conduction.*

It is based upon the principle that in disease or obstruction of the conduction apparatus, bone conduction is increased; while in disease of the auditory nerve or labyrinth, bone conduction is diminished.

The test is made by placing the tuning-fork (preferably the 256 d. v.) in firm contact with the median line of the head. The fork may be placed on the vertex, forehead, teeth, or chin. The patient is asked to notice with which ear he hears the tuning-fork loudest. If he has disease of the middle ear or an obstruction in the external auditory canal, bone conduction will be increased on that side, while the opposite side remains unaffected. If, then, the patient complains of deafness in the right ear, and the Weber test shows that he hears the fork best on that side, it is fair to presume that he has disease of the conduction apparatus rather than labyrinthine disease. If, however, he hears the fork best in the normal ear, it is a fair presumption that he is affected with labyrinthine or nerve deafness. Judgment should be reserved, however, until all tests and diagnostic signs are noted.

The Rinné Test.—This test is for the purpose of determining the *relative hearing power by bone and air conduction.*

Normally, the forks should be heard from fifteen to twenty seconds, longer by air conduction than by bone conduction; for example, if fork *a′* (435 d. v. Edelmann) is placed against the mastoid process and held there until the patient ceases to hear it, and is then held close to the ear, he should continue to hear it for fifteen to twenty seconds more.

If he has middle-ear disease, bone conduction will be increased; hence he will hear it longer than normally over

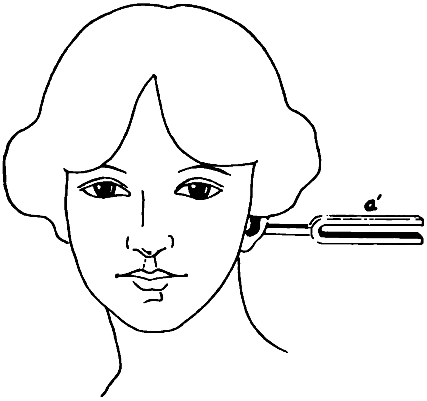

FIG. 47.—Showing the a' fork in position on the mastoid process for testing the bone conduction in the Rinné test.

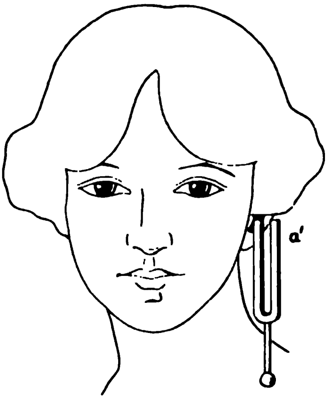

FIG. 48.—Showing a' fork held close to the ear for testing the air conduction in the Rinné test.

the mastoid process, which will leave a shorter time for it to be heard by air conduction.

If, however, there is labyrinthine disease, both bone and air conduction will be diminished or totally lost. As the conduction apparatus is normal, the sound will be transmitted to the labyrinth in a normal manner, but the perception of the sound will be impaired for both the bone and air conduction.

In middle-ear deafness of marked degree the hearing by bone conduction may be increased to such an extent that the fork will be heard longer over the mastoid than by air conduction. In this case the test should begin by holding the fork near the affected ear until it ceases to be heard, and then placing it against the mastoid process, note being taken of the number of seconds it continues to be heard in this position.

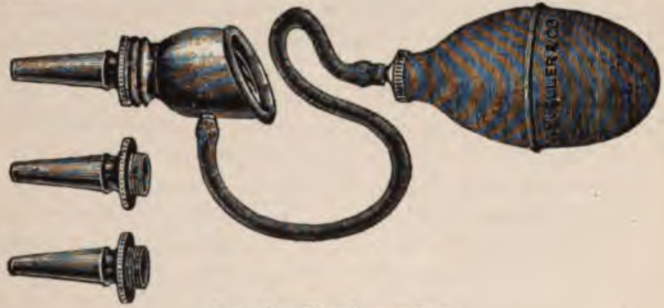

FIG. 49.—Siegel speculum.

The Gellé Test.—This test was devised by Gellé to determine the presence or absence of an ankylosed stapes. If air is compressed in the external canal of a normal ear by means of an air-bag or Siegel speculum, the tone of a vibrating tuning-fork placed on the vertex will be perceived greatly diminished. If ankylosis is present, the pressure will not be transmitted to the labyrinthine fluid, consequently there will be no diminished tone.

Principles Underlying the Tests of Hearing.—1. Normal range of hearing is from 16 to 22,097 double vibrations per second.

2. When the conduction apparatus is diseased or obstructed, the power to hear low tones is impaired or lost.

3. When the perception apparatus is diseased, the power to hear high tones is diminished or absent.

4. The normal ear hears the middle forks about twice as long by air conduction as by bone conduction. That is, a fork heard by bone conduction for twenty seconds, will be heard about forty seconds when held close to the ear.

5. When the conduction apparatus is diseased or obstructed, bone conduction is increased and the time left in which the fork should be heard by air conduction is diminished; or bone conduction may be so much increased that the fork is heard longer than by air conduction.

6. When the perception apparatus is diseased, both bone and air conduction are diminished or shortened, so that the relative time of hearing by air and bone may be the same.

Referring to the *third principle*, we note that when the perception apparatus is diseased the power to hear high tones is diminished or lost; and we note, in *principle five*, that hearing by bone conduction is increased. When we find deafness in a given ear with loss of hearing power for high tones and diminished bone conduction in the same ear, it is safe to presume the deafness to be due to labyrinthine, nerve, or auditory centre disease. If, however, a patient presents himself with deafness in a given ear, and the Weber test shows bone conduction increased on that side, and the Rinné test shows that hearing by bone conduction is as long or longer than by air conduction, and that the lower tuning-forks are not heard, while the upper forks and Galton whistle are heard distinctly, it is fair to presume that the case is one of middle-ear disease or external auditory canal obstruction.

The normal range of hearing for musical tones being from 16 to 22,097 double vibrations per second, it therefore follows that any marked deviation from this rule is a sign of some abnormality in either the conduction apparatus (middle ear and external ear) or the perception apparatus (labyrinth, auditory nerve, or auditory centre in the brain).

Bone conduction is frequently diminished in persons more than fifty years of age. This should be taken into account in estimating the value of the Weber and the Rinné tests.

Many cases in actual practice have at the same time disease of the external, middle, and internal ears, thereby modifying perception by both bone and air conduction.

Bone conduction may be increased by the presence of a plug of cerumen more than it is decreased by increased tension of the labyrinthine fluid from pressure on the foot plate of the stapes, or congestion of the labyrinth, so that the Rinné test may show a relative increase of bone conduction over air conduction. Thus the disease of the internal ear is masked. After the removal of the plug of cerumen, bone conduction is diminished below the normal limit, thus unmasking or revealing the internal-ear disease.

In suppurative disease of the middle ear there is often an increase in bone conduction to such an extent as to exceed air conduction; hence when internal-ear disease is combined with it, the influence of the internal-ear disease may predominate and show diminished bone conduction, which fact, if taken alone, would not call attention to the coexisting disease of the tympanum.

Boiler-makers, artillerymen, etc., frequently suffer from impairment of the nerve endings in the labyrinth; hence have diminished bone conduction. They may at the same time suffer from pronounced middle-ear disease, whereby bone conduction is increased, thus again bringing the conflicting signs in opposition.

When the physiologic tests, history, course, and symptomatology are given due consideration, the apparent contradictions will become clear and intelligible, and a correct diagnosis be made.

ETIOLOGY OF EAR DISEASES.

The factors concerned in producing diseases of the ear may be divided into predisposing and exciting.

Predisposing.—**Age.**—Children are more frequently subjected to the acute serous and suppurative diseases of the ear than are adults. This is due to two reasons chiefly:

1. Children are frequent victims of acute rhinitis and acute tonsillitis with the consequent hypertrophy of the nasopharyngeal lymphatic tissue.

2. In infants and children the Eustachian tube is relatively shorter, straighter and of greater diameter than in adults, and also more nearly approaches the horizontal plane, hence

infection spreads more easily from the nasopharynx to the ear.

Occupation.—(1) Exposure to all kinds of weather predispose to nose and throat trouble and this in turn affects the middle ear through the Eustachian tube. (2) In the same manner those occupations of a dusty nature such as street sweepers affect the ear. (3) Occupations which subject the ear to frequent loud noises such as boiler-makers, artillery-men, steel-workers, etc., produce a degenerative condition of the auditory nerve endings. (4) Sudden or prolonged changes in air pressure such as occur in caisson-workers, glass-blowers, cornet-players, etc., frequently lead to tympanic disturbances. (5) Drugs such as alcohol, quinine, salicylates, sometimes cause a chronic congestion of the auditory nerve which may later lead to nerve degeneration. Heredity is a more important factor in the production of diseases of the internal than of the middle ear. Certain cases of deaf-mutism and otosclerosis are good examples of this class.

Nasal Obstruction.—Nasal obstruction is a most important predisposing factor in middle-ear disease. Some of the common conditions producing this nasal stenosis are adenoids, deviated septum, hypertrophic rhinitis, turgescent rhinitis, polypi, etc.

Exciting Causes.—1. **Acute Nasopharyngeal Infection.**—Acute nasopharyngeal infection as manifested by acute rhinitis or "cold in the head" and by acute tonsillitis.

2. **Chronic Nasopharyngeal Infection.**—Chronic nasopharyngeal infection which may take the form of chronic rhinitis, pharyngitis, or tonsillitis.

3. **Acute Infectious Diseases.**—The acute exanthemata that are most frequently complicated by middle-ear infection are: measles, scarlatina, diphtheria, whooping-cough, and influenza. Pneumonia often leads to middle-ear suppuration. However, it should be borne in mind that the greater percentage of middle-ear complications of these acute infectious diseases occur in children harboring a nasopharyngeal hypertrophy.

4. **The Nasal Douche.**—The nasal douche, if improperly administered or if strangling or coughing occurs during the

procedure, will frequently force infection into the Eustachian tube or middle ear.

5. **Swimming or Diving.**—Swimming or diving may also produce an acute attack of otitis by forcing the water into the Eustachian tube or by water gaining entrance into the middle ear through a perforated drum membrane.

THE AURICLE.

CONGENITAL DEFECTS OF THE AURICLE.

There may be an entire absence, a partial development (microtia), or a misplacement of the auricle. In rare instances

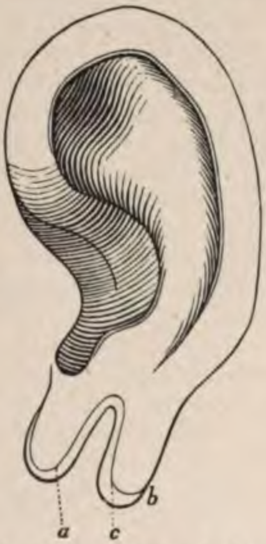

Fig. 50.—Cleavage of the lobule with edges freshened for surgical reunion.

it has been located on the cheek. Occasionally there is a blind canal, known as the *fistula auris congenita*, located in the ascending part of the helix and opening either just anterior to the tragus or upon its inner surface at the entrance of the external meatus. It can be obliterated by laying it open with a knife and removing the secreting membrane. Very rarely there is a supernumerary (polyotia) auricle.

INJURIES TO THE AURICLE.

These may consist of cuts, bruises, burns, cleavage of the lobule, sloughing, etc.

Hematoma.—Hematoma of the auricle (othematoma), may arise from a blow in football-playing, boxing, etc. It may also be spontaneous.

In slight cases cooling lotions should be applied. In the severer cases the tumor should be incised and thoroughly freed from clots and other detritus, after which it should be thoroughly irrigated and sutured, except at its most dependent portion, into which a small wick should be inserted for drainage.

Frost-bite.—When the auricle is frost-bitten, it becomes yellowish-white in color, and in extreme cases may be actually congealed and brittle. The treatment should consist in the immediate application of cold and friction for the restoration of the circulation. After this has been accomplished, the local application of heat and of the lead-and-opium wash should be made. If there are bluish or inflamed areas beginning to ulcerate, applications of the tincture of iodine or collodion act favorably. If gangrene or sloughing sets in, it should be treated with boric acid or iodoform ointments.

Perichondritis.—Perichondritis of the auricle is characterized by rapid development, the skin being hot, red or purplish in color, and painful. Upon incising the swollen parts there is frequently a discharge of fluid or pus, and the probe shows the perichondrium to be detached. The exposed cartilage presents a roughened surface when the probe is passed over it. The skin is infiltrated, the swelling often extending into the external meatus, thereby partially or completely occluding its caliber. The disease runs a more acute course than hematoma, and the history of traumatism may be absent.

When treated early, the application of cold and the lead-and-opium wash often checks the progress of the process. In the later stages, when fluctuation appears, it should be incised, irrigated, and closed with suture and a bandage applied. Healing usually occurs by first intention if there is a thorough removal of all morbid material and antiseptic precautions are observed.

Cysts.—Cysts of non-inflammatory origin, containing clear fluid, sometimes form on the auricle.

DISEASES OF THE AURICLE.

Eczema.—Eczema of the auricle usually occurs in connection with eczema of the external auditory canal, and will be further considered in that connection.

Lupus.—Lupus of the auricle occurs in connection with lupus of the skin of neighboring parts.

Syphilitic Inflammation.—Syphilitic inflammation of the auricle is one of many associated expressions of the disease, and should be treated according to the usual indications.

Hyperemia.—Hyperemia of the auricle may follow frost-bite, hot poultices, eczema, and inflammatory ear diseases. It also occurs in neurotic patients and in valvular disease of the heart.

Diffused Inflammations.—Diffused inflammations of the auricle may occur as the result of injury, frost-bite, heat, stings, and infections.

THE EXTERNAL AUDITORY CANAL.

CONGENITAL MALFORMATIONS AND INJURIES.

Congenital malformations, as dilatation, atresia, entire absence, and double canal are occasionally observed.

Injuries to the external auditory meatus may result from the accidental or intentional introduction of instruments and other hard bodies. Persons affected with "itching ears" often attempt to relieve the disagreeable sensation by scratching the external meatus with a pin or other hard instrument, and sometimes injure the skin in the attempt. Live insects occasionally gain entrance into the meatus, and unskilful attempts to remove them often result in injury. Injuries may also arise from the introduction of hot water, caustic fluids, and the unskilful use of the knife in paracentesis of the drum-head. Hot cinders also occasionally lodge in the meatus and excite inflammation.

Ceruminal Accumulation or Retention.—The plugs are usually composed of an admixture of *cerumen*, *sebaceous material*, *epidermis*, and *short hairs*.

Etiology.—Recurring hyperemia of the external meatus leads to the retention of cerumen. Contraction of the meatus and tenacious secretion also favor its retention. The ceruminal glands are located in the cartilaginous portion of the meatus, and the secretion is usually spontaneously discharged. Attempts to remove it with the twisted end of a towel forces the cerumen into the osseous portion of the meatus, where it becomes inspissated and admixed with epithelium and short hairs, and finally completely fills that portion of the canal. Eczema and other inflammations of the external auditory meatus favor ceruminal accumulations. Foreign bodies lodging in the canal act as foci around which the secretion and epithelium gather.

Symptoms.—There may be a feeling of fulness and confusion, with tinnitus. The patient's voice seems very resonant to himself, and he sometimes complains of giddiness and vertigo. In very rare cases there are vomiting and epileptiform convulsions. There is usually dulness of hearing, although when associated with perforation of the drum-head the plug may improve the hearing by acting as an artificial membrane. Conversation is usually understood by the patient. When speech is not understood, there is probably an associated middle- or internal-ear disease. Frequently a dry reflex cough is due to an impacted mass of cerumen. The ceruminal plugs appear as light yellow or dark brown, dull masses. They may be soft to the point of liquefaction or as hard as stone.

Prognosis.—Where the plugs are primary or unassociated with disease of the middle and internal ears, the prognosis as to hearing is good.

Treatment.—The instrumental removal of the cerumem is sometimes attended by considerable pain, and there is danger of injuring the skin of the meatus or forcing the plug against the drum-head, thereby occasioning great distress. The syringe affords an easy and safe means for its removal. A syringe holding from three to four ounces is best adapted to the purpose. If after several forcible injections of water the cerumen is not removed, the ear should be dried and the attempt discontinued. The patient should be given a phial of glycerin and instructed to instil it into the ear four times daily for two days. At the end of this time he should return to the office and the warm-water injections with the syringe be resumed. The instillations of the glycerin will have softened the cerumen, so that it may be easily removed with the syringe. After the removal of the ceruminal plug the external meatus should be painted with carbolized lanolin or other antiseptic ointment.

Absence of Cerumen in the External Meatus.—This condition is often observed after a diffused or circumscribed inflammation of the external ear, and in the course of adhesive processes in the middle ear. The trophic nerves seem to be affected by these diseases, thereby interfering with the functional activity of the ceruminal glands.

Treatment.—The treatment consists of mercurial ointments applied at intervals of forty-eight hours and the internal administration of nux vomica.

DISEASES OF THE EXTERNAL AUDITORY CANAL.

Furunculosis.—**Synonyms.**—Follicular Inflammation; Circumscribed Inflammation and Boils.

Occurrence.—Furunculosis of the external auditory canal is limited to the cartilaginous portion. The boils may be localized or associated with general furunculosis.

Etiology.—They may arise from frequent syringing of the ear, or from attempts at the instrumental removal of cerumen or foreign bodies. Streptococcus and Staphylococcus pyogenes frequently enter the deeper layers of skin through the hair follicles and cause the inflammation. Anemia and disturbances of menstruation and digestion, and exposure to cold air, also favor its appearance.

Symptoms.—If the boils are located in the superficial layers of the skin, the pain is slight; while if in the deep layers, it may be quite severe. There are often tenderness and swelling over the mastoid region, which may cause the condition to be mistaken for acute *mastoiditis*. The hearing is more or less impaired and tinnitus may or may not be present. There is often an itching sensation in the meatus, and the patient has an irresistable desire to use a pin for its relief. There is usually a slight febrile movement, especially at night. If the circumscribed inflammation is deep, it appears as a flat, red elevation; if superficial, it is circumscribed, pointed, red or livid, and attended with but slight pain. The pus may discharge spontaneously after eight or ten days. The boils are usually located on the anterior cartilaginous wall. Movement of the pinna causes severe pain, whereas in mastoiditis the pain is absent or very slight.

Treatment.—In the deep-seated type free incision should be made to relieve the pain. It is not well to wait for pus to form, as an early incision is often followed by improvement. If the incision is made after the formation of pus, slight pressure should be applied with a probe to assist in the removal of the tenacious pus from the deeper tissues.

In the *early stages*, while there are considerable irritation and pain, the application of cold with the Leiter coil will modify the progress of the disease. For the relief of pain the instillation of a 10 per cent. phenol-and-glycerin solution should be used. Hot fomentations applied at frequent intervals is also a useful remedy.

After the boils have discharged their contents spontaneously, or by incision, they may be treated by the remedies mentioned, after the application of which the meatus should be thoroughly dried and packed with a narrow strip of gauze. This should not be removed by the patient, but should be left in place and removed by the physician on the following day. The same process should be repeated until the inflammation has subsided. In cases in which there are recurrent attacks of furunculosis there should be appropriate constitutional treatment. Autogenous vaccines are frequently of service.

Diffuse Inflammation.—**Etiology.**—This type of inflammation may be due to the instillation of irritating drugs, injuries, foreign bodies, bathing in infected water, and associated mastoid disease.

Symptoms.—Pain is present in varying intensity. It is worse at night and upon movements of the jaw, or fits of coughing. Tinnitus and giddiness are sometimes present. The hearing is normal, except in those cases in which the canal is completely closed. The osseous portion of the canal is hyperemic. After two or three days there may be a discharge, which becomes purulent as the disease advances.

Treatment. — In cases due to traumatism the application of the Leiter coil and local asepsis, with gauze packing, is usually followed by a favorable termination. If the disease has advanced to the stage of secretion, antiseptic powders and a saturated solution of boric acid in alcohol should be used. In very obstinate cases, especially when granulations are present, a strong solution of silver nitrate applied locally reduces the granulations and promotes epidermization. Sometimes great relief is obtained by incision at the point of greatest tenderness.

Otomycosis; Mycosis.—This is a *parasitic inflammation* due to the Aspergillus niger, A. flavus, and A. fumigatus. Occasionally other fungi are also present.

The growths *appear* as brownish or black plaques upon the skin of the meatus, or upon moist crusts.

When growing upon moist crusts, they may form layer upon layer, until the meatus is more or less filled. They show a tendency to recur at intervals of days or weeks. In doubtful cases the microscope will aid in the diagnosis.

Treatment.—Treatment consists in the instillation of alcohol, which stops their growth after one or two applications. The treatment should be repeated frequently for a year to prevent their return.

Eczema of the External Ear.—Eczema in this locality differs in no way from eczema on other portions of the skin. It may be *acute* or *chronic*, but is more often of the former type. It may attack all ages and either sex, but is more common in children. It may be *erythematous*, *vesicular*, *pustular*, or *squamous*. It may be limited to the auricle or to the external auditory canal, or it may involve both at the same time.

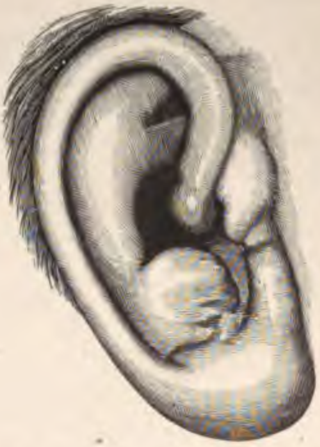

Fig. 51.—Thickening and deformity of the auricle, due to chronic eczema. (Bacon.)

Symptoms.—If of the *acute type*, the skin is infiltrated and swollen, and may obstruct the external auditory meatus.

If it is of the *chronic type*, there is permanent thickening of the tissue, which may occasion considerable deformity of the ear (Fig. 51).

A feeling of fulness, moderate deafness, and tinnitus are often complained of.

Treatment.—Constitutional dyscrasiæ should be corrected by the administration of the proper remedies. In the local treatment, the use of water should be avoided, as it aggravates the condition. If it is of the moist variety, the eczematous areas should be painted with a 10- to

30-grain solution of silver nitrate. If it is of the dry, scaly type, an ointment containing 1 grain of salicylic acid and 4 minims of the tincture of benzoin in 1 dram of lanolin, applied twice daily, is followed by great relief from the stiffness and excessive epithelial desquamation. After removal of the crusts, the meatus should be powdered with equal parts of boric acid and zinc oxide. A 10 per cent. ichthyol or resorcin ointment, locally applied, is also a very useful remedy. Moist surfaces back of the auricle, especially at the union of the auricle and the skin over the mastoid, should be dusted with borated powder or with pulverized calomel.

Seborrhea.—This condition is usually associated with a similar affection of the face and scalp.

Symptoms.—When it affects the external auditory meatus, there are itching, tinnitus, and slight deafness.

Treatment.—The treatment should consist in removal of the scales, after which the parts should be dusted with equal parts of boric acid and zinc oxide. An ointment of white precipitate is also a useful remedy.

Foreign Bodies in the Canal.—Insects, such as cockroaches, bed-bugs, fleas, beetles, etc., sometimes get into the external canal, and give rise to very distressing symptoms. Wads of cotton, paper, portions of pencils, seeds, beads, cherry-stones, and many other inanimate objects have also been found. Beans, wheat, and other grains, which swell upon being exposed to moisture, may give rise to marked *pressure symptoms.*

Symptoms.—Symptoms in general are those of obstruction to the meatus, attended by more or less inflammation.

Treatment.—Do not attempt to remove live bugs from the ear, as the effort may prove fruitless, and occasion great distress to the patient. They should first be drowned in water, oil, glycerin, or alcohol, after which they can be easily removed with the syringe. If this method fails, they may be removed with forceps, or a short-toothed ear hook. A syringe (Fig. 52) with a flat, thin nozzle, which can be inserted at the side of the foreign body may be used to force water back of it, and thereby drive it outward. Smooth, hard bodies, like beads, may be removed by dipping a camel-hair brush in glue and applying it to the surface of the foreign body, leaving it there until it becomes quite dry. The removal of the brush will bring the foreign body with it.

Ear-cough.—Foreign bodies and inspissated cerumen may excite an irritation of the auricular branch of the pneumogastric nerve which supplies the external canal.

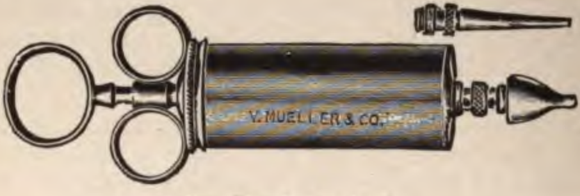
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Fig. 52.—Ear syringe.

Stricture of the Canal.—**Etiology.**—Stricture of the canal may be due to swelling of the skin in chronic suppurative otitis media, and other persistent inflammations and irritations. *Bony stricture* (hyperostosis) may follow caries and exfoliation of the sequestra from the osseous walls of the canal. Septa or adhesions may form across the canal by the approximation of granulations or inflamed surfaces.

Treatment.—When the stricture is due to thickening of the soft tissues of the meatus, it may be dilated with bougies. If this method fails, longitudinal incisions should be made in the meatus and cotton plugs or sponge tents applied. If adhesions or septa are present, they should be divided and distended with cotton plugs or sponge tents.

Exostosis of the Canal.—This is a circumscribed bony growth projecting from the wall of the osseous portion of the external auditory canal. It is more common in males than females.

Treatment.—Treatment consists in removal with the cold-wire snare, the dental burr or chisel. It may also be removed by making a postauricular incision at the junction of the auricle with the mastoid, and pulling the auricle and soft meatus forward, as for a mastoid operation. The tumor is thus fully exposed, and may be removed with a chisel or a dental burr. The bone beneath it should also be slightly excavated so as to prevent recurrence.

Injury to the Drum-head.—**Etiology.**—Rupture of the drum-head may be caused (*a*) by direct violence, as from some hard penetrating body; (*b*) by injury to the bones of the cranium, as from a blow on the chin; and (*c*) by concussion of the air, as following the firing of a cannon or a blow with the palm of the hand on the auricle.

Symptoms.—The rupture is usually in front of the handle of the malleus. The drum membrane is covered with a bloody discharge. If infection has not been introduced, the rupture usually heals quickly. The labyrinth may be involved. The bone-conduction and Galton-whistle tests should be used to determine this fact. When it is involved, there are usually giddiness, nausea, and vomiting.

Treatment.—The treatment consists in the insertion of a strip of dry sterilized gauze into the external meatus.

Acute Myringitis.—**Symptoms.**—In the early stage it is characterized by redness of the drum-head in the posterior quadrant, or it may involve the whole surface. The inflammation is often limited to the dermic layer, although it may extend through the fibrous to the mucous layer. It is in such cases that pus pockets form between the fibrous and mucous layers. The drum-head presents much the same appearance as it does in suppurative disease of the middle ear. When the dermic layer is involved, vesicles filled with serum form, and voluntarily discharge their contents in eight to ten hours. At the onset of the attack there is a sharp, stinging pain, radiating to various parts of the head. In the *superficial type* it soon subsides, while in the deeper variety it may exist for some time. There is a sense of pressure, fulness, tinnitus, and moderate deafness. There may be hyperesthesia to noises. The disease runs its course in from three to five days.

Treatment.—If there is marked inflammatory action, the application of the Leiter coil or the artificial leech in front of the tragus exerts a favorable influence upon the course of the disease. The serous vesicles should be punctured, care being exercised to avoid penetrating the deeper layers. If there is an abscess in the deep layers, it should be freely incised to prevent the spontaneous discharge of pus into the middle ear. In doing this, extreme care should be used to avoid opening through the mucous layer. Should the mucous layer be incised, the pus and germs will enter the middle ear and excite a similar process there.

Chronic Myringitis.—Etiology.—This form of inflammation is rare, and involves the whole surface of the drum-head. It may be due to repeated attacks of acute myringitis. It sometimes follows diffused or follicular inflammation of the canal and is often associated with chronic middle-ear suppuration which has been followed by closure of the perforation.

Symptoms.—The membrane has a moist, lustrous appearance, is grayish in color, with yellowish spots here and there upon its surface. The handle of the malleus is injected. At times the dermic layer of the drum-head is thrown off, after which the membrane beneath is more smooth, red, and velvety. In some cases small papillæ form, giving the surface of the membrane the appearance of a raspberry.

The disease is usually unattended by pain or other marked *subjective* signs. There is slight itching, and in those cases in which there is rapid exfoliation of the dermic layer, followed by a serous exudate, there may be an offensive odor. This is due to the retention and decomposition of the epithelial cells and exudate.

Prognosis.—The drum-head is rarely perforated. When there is a cessation of the serous exudate, recovery occurs spontaneously. Whether cure is effected spontaneously or through treatment, there is a moderate thickening of the drum-head and slight impairment of hearing, although the process may have existed for several years.

Treatment.—In mild cases, antiseptic irrigation, followed by mopping the parts dry and dusting with boric acid powder, often causes a cessation of the secretion and restores the parts to their normal condition. In the more obstinate cases the canal should be irrigated and a saturated alcoholic solution of boric acid instilled for ten to fifteen minutes, three times a week. When granulations or papillæ form upon the drum-head, they should be painted two or three times a week with a 2 to 10 per cent. silver nitrate solution.

DISEASES OF THE MIDDLE EAR.

Acute Tubal Catarrh.—**Synonym.**—Acute tubotympanic catarrh.

Etiology.—The most frequent cause of tubal catarrh is some pathological condition of the nose and throat of which the adenoids are probably the most common. Frequent attacks of coryza and acute nasopharyngitis causes an associated swelling and closure of the Eustachian tube. Vigorous blowing of an obstructed nasal passage may force some infected mucous into the Eustachian tube with the resultant swelling of the mucosa and closure of the tube.

Pathology.—This is chiefly a hyperemia and swelling of the mucosa of the Eustachian tube, with at times an associated congestion of the mucosa of the middle ear. If the closure of the tube persists, the air within the tympanic cavity is more or less absorbed and an increased hyperemia of the middle ear results. This congestion of the middle-ear mucosa produces an increased serous or mucous transudate.

As the air within the tympanic cavity becomes rarefied, the drum membrane is drawn in and the condition known as *retraction of the drum membrane* is produced. This retraction is characterized by certain changes in the appearance of the membrane as viewed by reflected light, namely: (1) Prominent short process due to the drawing inward of the handle of the malleus and the resultant protruding outward of the short process. (2) The drawing inward of the handle of the malleus produces an apparent shortening called foreshortening of the handle of the malleus. (3) The light reflex is broken, misplaced, or absent. (4) Frequently the drum membrane is red and shiny, similar to the appearance of waxed paper over red cloth. The handle of the malleus looks white and thicker than normal.

Symptoms.—If the pathological process is confined to the Eustachian tube, the symptoms are very slight. The patient complains of a stuffiness or sense of fulness in the ears, some pain described as a dull ache may be complained of, especially if the middle-ear cavity is greatly congested. Autophonia, or resonance of the patient's own voice is an annoying symptom which occurs in many cases. Frequently all symptoms are absent, except when the patient stoops or leans forward, when all or part of the above symptoms are noticed.

Diagnosis.—This is made from the above symptoms, from the abnormal position of the drum membrane and from the presence of an exudate within the Eustachian tube or within the middle-ear cavity. The presence of an exudate is determined by the bubbling or snapping sounds that are heard during a catheter inflation.

The functional examination of the ears during an attack would reveal the Weber localizing on the affected or more seriously affected side. The Rinné would show a slight lowering for air conduction, and a slight increase in bone conduction. The low limit would be somewhat raised and the high limit would be normal.

Prognosis. Good for the immediate attack with proper treatment, but if the etiological factors are not removed recurrences and finally a chronic condition ensues.

Treatment. This consists in the restoration of the normal patency of the Eustachian tube, the drainage of the exudate, and the removal of the etiological factors.

The Eustachian tube is best restored to its normal patency in adults by means of the Eustachian catheter. In children this can be accomplished more easily by means of the Politzer bag (for technic of tympanic inflation, see page 227). Every second day the inflations should be given. The tube is thus opened, air forced into the tympanum, and the fluid allowed to flow out. The drum-head and malleus are thus pushed outward and made to assume their normal positions, and incipient adhesive bands are broken up. Free catharsis helps in depleting the tissue and should be given in all cases. Hot gargles may be of some assistance in relaxing the pharyngeal tissue. The nose and nasopharynx should receive appropriate cleansing, antiseptic treatment either by spraying with liquor alkalinus antisepticus, Dobell's solution, saturated solution of boric acid, etc., or by the application with swabs of

℞—Iodine		gr. xv
Potassium iodide		gr. xx
Oil gaultheria		♏v
Glycerin	q. s. ad	ʒj

If the foregoing treatment, after continuing for ten days to two weeks, does not give relief and the exudate continues to reform to the extent of bulging in certain portions of the membrana tympani, then it will be necessary to resort to *paracentesis* or incision of the drum membrane.

Technic of Paracentesis.—1. The auricle and external canal is sterilized by painting with tincture of iodin and alcohol equal parts or by scrubbing with soap which is allowed to dry thoroughly.

2. The instruments and hands of the operator are sterilized. The instruments used besides the head mirror and light are, ear speculum, cotton carrier, ear forceps, medicine dropper, and a paracentesis knife.

3. The anesthetic may be general or local. With children it is advisable to administer a general anesthetic as the shock and fright from the operation in susceptible cases may lead to certain nervous disorders such as chorea or hysteria, etc. In adults a local anesthetic will suffice. Twelve per cent. phenol in glycerin, if left in contact with the ear drum for five or ten minutes, is very satisfactory. A combination suggested by Grey is:

℞—Cocain	gr. xv
Anilin oil	ʒj
Alcohol	ʒj

4. The incision (Fig. 53) is made with a sharp-pointed paracentesis knife, beginning in the upper portion and cutting downward and slightly forward in a curved line. Having completed the incision, the ear should be catherized or Politzerized in order that the retained fluid of the middle ear may be forced through the incision into the external canal.

Acute Catarrhal Otitis Media.—**Etiology.**—Same as of acute tubal catarrh.

Pathology.—The pathological process involved in this disease is essentially a continuation of that of acute tubal catarrh. The mucous membrane of the Eustachian tube and the middle-ear cavity become swollen and hyperemic. There is an exudate of thin serum or of thick mucus or a combination of the two. This exudate may completely fill the tympanic cavity. More or less desquamation of the mucous membrane occurs.

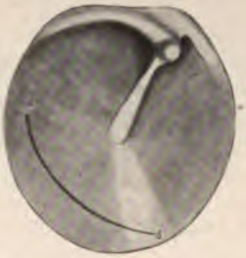

FIG. 53.—*A*, *B*, the line of incision for the evacuation of mucous or serous exudate.

Symptoms.—The patient may first notice a stuffiness or sense of fulness in the ear with some tinnitus and deafness. As the pressure within the tympanic cavity increases, a dull ache rapidly changing to a sharp pain is complained of. The temperature is normal or only slightly above normal. If the tympanic membrane is examined when the sharp pains are complained of, all the features of a normal drum membrane will have disappeared. The membrane will be a bright red color. All the landmarks have disappeared. Bulging most frequently occurs in the upper and posterior portion.

If the disease is permitted to follow its own course, spontaneous perforation of the drum membrane may take place in which event the seromucous discharge is found in the external canal. Instead of escaping through the tympanic membrane the pent up secretions may escape through the Eustachian tube. In either event the symptoms are much relieved.

Prognosis.—This is good in the vast majority of cases if proper treatment is instituted early. However, some cases progress into the purulent stage and a suppurative otitis is instituted which may become chronic. In some cases a chronic catarrhal otitis persists.

Treatment.—In the early stages the treatment is the same as that given for acute tubal catarrh. If the pain is severe and some bulging of the drum membrane is present, paracentesis should be done without delay. This affords immediate relief for the pain. It is essential for rapid recovery that the discharge from the middle ear be drained away as rapidly as formed. This can best be accomplished by first wiping away the secretion with sterile cotton, mounted on small ear applicators and then placing a sterile gauze wick within the external auditory canal, so that one end of the wick is in contact with the perforation or incision of the membrane. This dressing should be changed daily or twice daily while the discharge is profuse. After the discharge has been reduced somewhat, the insufflation of a small amount of boric acid powder will frequently stop the discharge entirely. For the relief of the dull pain in the first stages, before drainage has been established, there is nothing better than a 5 to 10 per cent. solution of phenol in glycerin. The solution should be warmed and 5 to 10 drops applied to the ear every two hours.

Middle-ear Inflation.—**Purposes of Tympanic Inflation.**—Inflation of the middle ear is practised (*a*) to determine the permeability of the Eustachian tube; (*b*) to aid in arriving at a correct prognosis under treatment; (*c*) to determine the presence or absence of a perforation of the drum-head; (*d*) to clear the Eustachian tube and the middle ear of pus and secretions; (*e*) to restore a retracted drum-head to its normal position; (*f*) to restore normal intratympanic pressure and thereby relieve pain and engorgement; (*g*) to relieve undue pressure upon the foot plate of the stapes, and thus relieve intralabyrinthine pressure; (*h*) to prevent ankylosis of the ossicles; and (*i*) to break down newly formed adhesive bands.

Methods.—The three methods in most common use are (*a*) catheterization; (*b*) Politzerization; and (*c*) Valsalva's method of inflation.

Catherization of the Middle Ear.—*Instruments.*—This method is accomplished by the use of a silver or vulcanized rubber tube, suitably curved at one end, and having a funnel-shaped opening at the other for attachment to the Politzer bag, or to the shut-off of the compressed-air receiver.

The tubes made of soft or virgin silver are preferable, as they can be bent to suit individual cases. Those made of hard rubber can only be bent under hot water. They possess, however, two advantages over silver; namely, they are not affected by the fluids injected through them, and, as they are flexible they can be more easily insinuated around obstructions in the nares.

Preliminary to Catherization.—(*a*) Carefully examine the nares and postnares for obstructions and diseased conditions. (*b*) Cleanse the nares (anterior and posterior). (*c*) Cocainize the nasal and nasopharyngeal mucosa with cotton mops introduced through the anterior nares. This precaution will not only render the procedure painless, but will also contract the mucosa at the mouth of the Eustachian tubes, thus facilitating the entrance of air into the tympanum. One with delicate manipulative technic and large experience will rarely need to observe this precaution. (*d*) Pass a current of air through the catheter to free it from water or other obstructions. (*e*) Place one end of the *diagnostic tube* in the external meatus of the ear to be inflated and the other in your own ear. (*f*) Steady the patient's head with the left hand, the index and second fingers grasping the bridge of the nose, while the thumb elevates its tip.

Introduction of the Catheter.—*Politzer's Method.*—Direct the patient to close his mouth and swallow and keep the mouth closed, to relax the palatal muscles. The sterile and lubricated catheter is introduced with the right hand, the curved tip being turned downward and inward on the floor of the nose at its junction with the septum. It should be gently but quickly passed backward until it touches the posterior wall of the nasopharynx. It should then be turned outward and upward into Rosenmüller's fossa, in front of which is a rounded prominence forming the posterior wall of the mouth of the Eustachian tube. In other words, there is a prominence lying between the end of the catheter and the mouth of the Eustachian tube. The catheter should be held firmly, but with a *readiness to yield* to the contour of the surface over which it is to be drawn; with this intention in mind it is drawn forward over the prominence and then turned outward and upward into the depression or mouth of the Eustachian tube (Fig. 54).

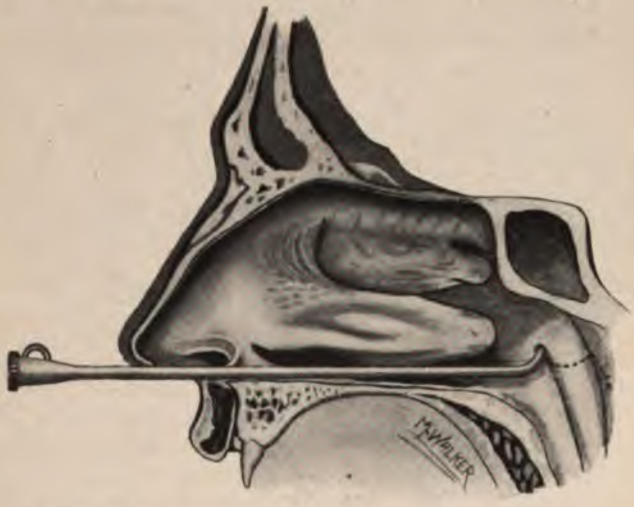

FIG. 54.—*X*, starting-point on posterior pharyngeal wall of tip of catheter and course it follows to enter the mouth of the Eustachian tube.

The eyelet upon the outer end of the catheter corresponds to the direction of the curved tip, and should point toward the auricle or outer canthus of the eye.

Gruber's method consists in introducing the catheter as described in the preceding paragraphs, and then withdrawing it, curved end downward, until the tip presses against the soft palate, when it should be rotated outward into the mouth of the tube.

Lowenberg's method consists in turning the curved end of the catheter inward toward the opposite side and withdrawing it until it engages the posterior end of the septum. It is then rotated downward and outward, describing a half-circle, into the mouth of the Eustachian tube.

This and the *Politzer* method are the most reliable, as they have fixed anatomical guides—*i. e.*, the posterior end of the septum and Rosenmüller's fossa; while Gruber's plan depends upon the soft tissue. When one method fails, the other should be tried and will usually be attended with success.

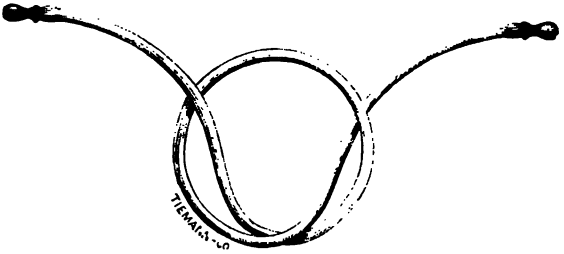

Fig. 55.—Auscultation tube.

After Introduction of the Catheter.—Attach the *Politzer bag* or the *shut-off* of the compressed-air chamber to the outer end of the catheter and force air through it into the Eustachian tube and middle ear. By means of the *diagnostic tube* (Fig. 55) the operator may know whether or not air is entering the middle ear. There will be a soft blowing or bubbling sound, if it is properly gaining admission. The membrana tympani may sometimes be heard to snap as it returns to the normal position. After several interrupted blasts, the catheter should be removed, first turning the curved tip downward.

Dangers Attending the Use of the Catheter.—The mucous membrane may be atropic, or unskilful manipulation of the catheter may cause it to penetrate the epithelial covering of the mucosa. In either event inflation will be followed by emphysema of the submucous tissue of the neighboring parts, as the uvula, cheek, soft palate, or throat. A few cases have been reported in which there was a fatal issue from suffocation. The skilled physician need have little fear of producing such a result. The use of high pressure from a compressed-air tank is to be condemned on account of this danger. If the air tank is used, the gauge should not register more than forty pounds.

Politzerization.—*Instruments.*—This very useful procedure is done with a *soft-rubber bag* (Fig. 56), of about eight ounces capacity or by means of an air-pressure tank. To the hard-rubber fitting at the smaller end of the bag is attached a piece of *rubber tubing*, at the other end of which is a hard-rubber *nasal tip* for application to the anterior nares.

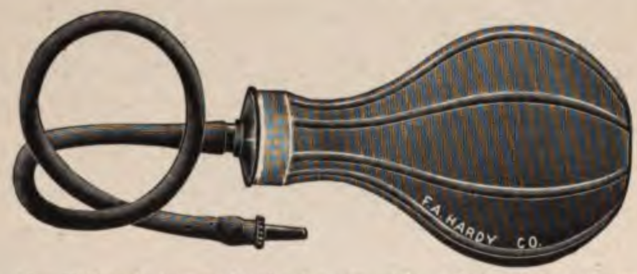

FIG. 56.—Politzer's bag and tube for use with a Eustachian catheter or nasal tip.

General Method.—The nasal tip should be inserted into one of the anterior nasal openings, the opposite nostril closed, and air forced into the nasal chambers by a sudden and forcible compression of the bag with the disengaged hand.

Unless certain precautions are observed, the air will escape into the pharynx and esophagus instead of going into the middle ear. There are *three methods* in common use which are more or less successful in rendering inflation by the Politzer method effective, namely: (*a*) Instruct the patient to forcibly inflate the cheeks, thereby causing the palatine arch to separate the nasopharynx from the lower pharynx. (*b*) Another method consists of pronouncing certain sounds, which momentarily shuts off the communication between the nasopharynx and pharynx, and at the same time opens the mouths of the Eustachian tubes. The sounds best adapted for this purpose are the English "k," or the German "och."

At the moment of pronunciation the bag should be foricbly compressed. (*c*) The third and the most successful method requires the patient to take a sip of water in the mouth and retain it there until told to "swallow," and at the height of the act of swallowing the physician compresses the bag as in the other methods.

By the second and third methods air is shut off from the lower air passages, the mouths of the Eustachian tubes are opened, and air is allowed to pass into the middle-ear cavity. If, however, there is considerable thickening of the mucosa of the tubes, or if there is a *stricture* or fibrous band closing the tubes, it may be necessary to resort to the use of the catheter, and in extreme cases to the Eustachian bougies.

Valsalva's Method of Inflation.—This method is mentioned chiefly for the purpose of condemning it. It is performed entirely by the patient, and therein lies the danger.

Method.—The patient should close both nostrils with the thumb and index finger, take a deep breath, and forcibly attempt to expel the air through the closed nostrils. The method is pernicious because of the ease with which the patient can resort to it, and because inflation when continued over a protracted period results in atrophy of the drum-head.

As a rule it is well to limit the inflations to a period of about six weeks. If good results do not follow in this time, it is useless to continue such treatment longer.

Acute Suppurative Otitis Media.—**Etiology.**—The same etiologic factors are involved in the production of an acute suppurative otitis media as in the production of an acute tubal catarrh, and as this has been discussed and a separate section has been devoted to the etiology of diseases of the ear, it will only be necessary to recapitulate the more common causes. (*a*) Catarrhal and suppurative conditions of the nose and nasopharynx. (*b*) Enlarged or diseased adenoid and tonsils. (*c*) Exposure to inclement weather. (*d*) Diving and injuries to the membrane. (*e*) Acute infectious diseases.

Pathology.—The first stages of an acute suppurative otitis media may be identical with the pathological conditions found in an acute catarrhal otitis media, namely, the congested and swollen mucous membrane of the Eustachian tube and middle-ear cavity with an outpouring of a seromucous discharge which soon becomes of a mucopurulent character. The mucous membrane becomes greatly thickened, which is most marked in the membranous folds of the attic. There is an extensive small-celled infiltration with more or less extravasation of red blood cells into the submucosa. As the inflammatory process advances and frequently quite early, perforation of the drum membrane occurs, generally in the inferior half, unless the infection is located in the attic, in which event perforation will probably appear in the membrana flaccida. In the most severe types of the disease necrosis of the ossicles and tympanic walls occurs.

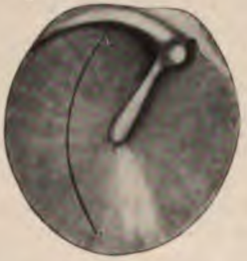

Fig. 57.—Right ear drum. *A*, *B*, the lines of incision in acute suppurative otitis media.

The infection may extend to the mastoid, to the brain, or to the venous channels located near the middle ear.

Bacteriology.—The bacteria commonly found in an acute suppurative otitis are streptococcus, staphylococcus, pneumococcus, Klebs-Loeffler, bacillus of influenza, and occasionally the tubercle bacillus. Secondary infections frequently occur and two or more of the above strains may be present.

Symptoms.—The symptoms are similar to those associated with an advanced acute catarrhal otitis media. The pain is more severe and is sometimes associated with tenderness over the mastoid antrum even prior to a mastoid infection. This is due to a coincident infection of the antrum. Tinnitus is present in nearly every case. This may persist for days even after the discharge has cleared up. More or less deafness in the affected ear is complained of.

A sudden rise in temperature to 102° or 103° F. with severe headache is usually present. In children the temperature may rise to 105° or 106° F. with evidence of marked constitutional involvement.

Treatment.—(See Treatment of Acute Mastoiditis.)

Rupture of the drum membrane or evacuation of the tympanic cavity through an incision in the membrane will cause marked lessening and in many cases a complete cessation of the symptoms with the exception of the mucopurulent discharge through the ruptured or incised drum membrane.

Acute Mastoiditis.—**Etiology.**—Acute mastoiditis is, in almost every instance, secondary to an acute suppurative otitis media or an acute exacerbation of a chronic suppurative otitis media. Theoretically it is possible to have a primary mastoiditis from trauma or from the blood stream transmitting syphilitic or tubercular infection. In actual practice, however, it is rarely seen.

Pathology.—The mastoid antrum is undoubtedly involved in almost all middle-ear suppurations, and when a virulent infection has occurred, or the free outflow of pus from the antrotympanic cavity is prevented, then the mastoid frequently becomes involved.

This extension of the infection to the mastoid process may occur in three ways:

1. Direct extension from the antrum as the result of pressure.
2. Gravitation of infected mucus or pus into the tip of the mastoid with an intervening healthy area.
3. Extension by means of the blood or lymph streams.

Three stages usually occur in the development of an acute mastoiditis: (1) hyperemia and cell infiltration; (2) suppuration; (3) necrosis of the bone.

Symptoms.—The symptoms may be divided into general and local.

The *general symptoms* consist of an elevation of temperature from 100° to 103° F., more marked in children than in adults and nearly always occurring in the first stages of the mastoiditis. At times fever is entirely absent, especially if good free drainage has been instituted from an early and large incision of the drum membrane. Direct extension of the infection from the antrum to the mastoid results in a lower temperature (frequently normal) than occurs from infection by gravitation or from infection carried by the vascular channels. The frequency of the pulse and respiration is increased in proportion to the fever. A chill is sometimes present during the onset of the disease, although not common. Headache and general physical unrest sometimes occur in the early stages. Occasionally one sees certain cerebral symptoms such as vomiting, vertigo and convulsions in the early stages. Sleeplessness is an important symptom and may occur even when there is no pain.

The *local symptoms* are more marked in the early part of the disease. In children there is more or less infiltration and swelling of the tissue covering the mastoid, so much so in fact that the ear may be displaced. The concha is pushed outward and downward. In this postauricular infiltration, movement of the auricle does not cause pain as it does in the auricular displacement and edema from furunculosis. Pain within the ear is generally a prominent symptom. It is usually deep-seated and boring, sometimes lancinating. Frequently the pain radiates upward over the side of the head.

The mastoid tenderness is an early and valuable sign. It varies from a dull ache or tenderness on firm pressure to a most lancinating pain from a slight touch. There are three points of greatest tenderness that can usually be determined. The most common point of tenderness is directly over the mastoid antrum at a point located just behind the attachment of the auricle and on a level with the superior wall of the external meatus (Fig. 61). The second point of tenderness is over the mastoid tip. The third point is over the emissary vein, located about one inch and a quarter behind the attachment of the auricle and on a level with the external auditory canal.

The appearance of the drum membrane before perforation and after very closely resembles that of a severe suppurative otitis media uncomplicated by a mastoiditis. Frequently

The application of cold in the form of an aural ice-bag or Leiter's coil in the early stages reduces the pain somewhat. A heavy rubber glove filled with cracked ice and tied at the top makes a very satisfactory substitute for an ice-bag. The cold applications should not be left on longer than twenty-four hours, as it may reduce the vitality of the parts. Heat applied to the mastoid gives more relief to some patients than cold and may be substituted for the ice-bag.

Morphine should not be given unless the pain is unendurable, as the narcotic may mask the symptoms.

The Indications for the Mastoid Operation.—In many cases of acute mastoiditis it is a debatable question whether to resort to operative interference or not; however, certain conditions that may arise are more or less universally accepted as indications for operation.

1. Auricular displacement due to the mastoiditis.
2. Persistent tenderness over the mastoid which is not relieved after five to ten days of free drainage through the drum membrane.
3. Persistent aural discharge which does not clear up after thorough non-operative treatment has been tried for six weeks.
4. Symptoms of vestibular irritation.
5. Meningeal irritation while the ear is still discharging.
6. Sagging of the posterosuperior canal wall which persists after free drainage has been established through the drum membrane.

The Simple Mastoid Operation.—Preparation of the Patient.—The operation should be done in a hospital.

Shave the head for two inches around the auricle, twenty-four to forty-eight hours prior to the operation. Scrub the skin with green soap and boiled water, after which it should be washed with alcohol and thoroughly dried. The external meatus should also be thoroughly irrigated and dried. Pack the meatus and cover the whole side of the head with sterilized gauze. These should not be removed until the patient is under the influence of the anesthetic, when the shaved area, pinna and external auditory canal are painted with tincture of iodin.

The patient should be placed upon a light diet, and be given a cathartic on the evening previous. He should take no food within twelve hours of the operation.

After anesthesia is well established, pin a sterilized towel or rubber cap around the patient's head and place it on a cushion to overcome the shock of the blows of the mallet.

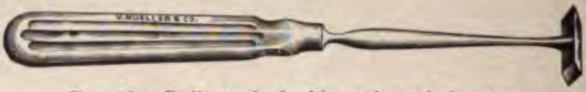

FIG. 58.—Ballenger's double periosteal elevator.

The wound should be well lighted during the operation. This may be done with a lamp and head mirror or by the use of an electric head lamp (Fig. 59).

The Postauricular Incision.—This should begin at the tip of the mastoid process, and follow, within one-quarter inch from the line of union between the auricle and the skin over the mastoid, to a point immediately over the superior auricular attachment. The tip of the mastoid should be located and the scalpel inserted through the skin and periosteum, cutting both as it follows the auricular attachment until the temporal muscle is reached, when the incision should divide the tissue down to the muscle but not through it. If the primary incision does not fully expose the operative field, a secondary

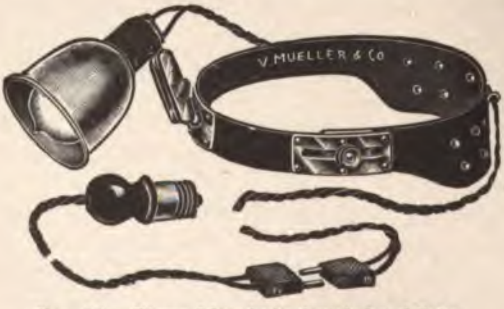

FIG. 59.—Pierce-Murphy's electric head light.

incision (Fig. 60) should be made at right angles to the primary one. The periosteum should be elevated or loosened with the periosteal elevator and the bleeding-points seized with artery forceps. The periosteum and skin of the post-superior wall of the meatus should also be separated from the bone with a small elevator. The ear is pulled forward and the posterior edge of the wound backward and held with retractors in the hands of assistants. A self-retaining double retractor is a better arrangement, as it leaves the assistant free to do other things. The tough tendinous

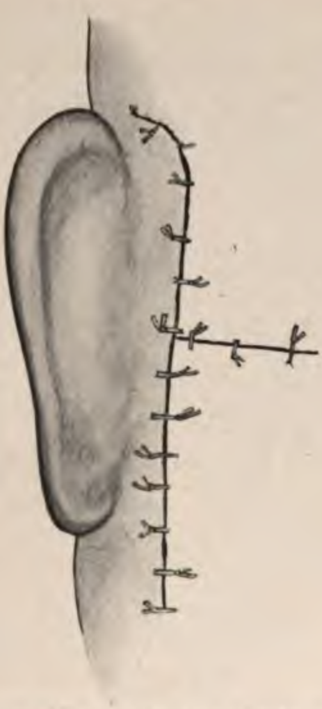

FIG. 60.—The secondary mastoid incision at right angles to the primary incision.

fibers of the sternomastoid muscle should be cut away from the tip of the mastoid by means of curved scissors.

The Anatomical Landmarks.—Having exposed the bone and meatus, certain landmarks should be sought as guides to the antrum by such a route as will avoid injury to the lateral sinus, the middle fossa of the cranium, and the facial nerve.

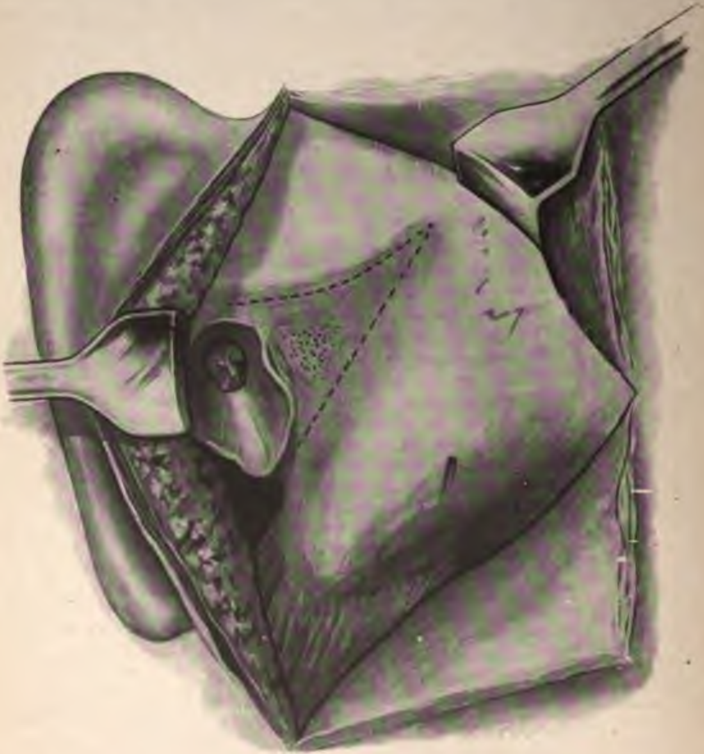

FIG. 61.—The anatomical landmarks for opening the mastoid antrum. The suprameatal triangle, the spine of Henle, and sieve-like depression.

By operating within the limits of the *suprameatal triangle* (Fig. 61), and following the direction of the external auditory canal to a depth of about three-eighths of an inch less than the depth of the bony meatus and middle ear combined, the sinus, cranial fossa, and facial nerve will not be injured, except rarely. The boundaries of the suprameatal triangle are as follows: (*a*) the anterosuperior boundary is formed by the posterior ridge or root of the zygoma; (*b*) the posterior boundary is an imaginary line extending from the posterior end of the zygomatic root, downward and slightly forward to the posterior tubercle of the external auditory process; (*c*) and the antero-inferior boundary is the curved margin of the postsuperior wall of the meatus. A fairly constant landmark to the mastoid antrum is the sieve-like perforations in the mastoid cortex just behind the external opening of the meatus (Fig. 61). Another landmark is the *suprameatal spine*, or the *spine of Henle*, a crescentic bony spine located on the upper, posterior margin of the external bony meatus.

An opening extending from the centre of the triangle inward in a direction parallel with the postsuperior wall of the external auditory canal will almost invariably expose the cavity of the antrum, unless it has been obliterated by sclerosis or other process.

The Removal of the Mastoid Cortex.—This may be removed in parallel shavings, as recommended by Whiting, or a section of the cortex may be removed in one piece. The gouge should not enter the cortex at right angles, but should be directed obliquely, thereby avoiding injury to the sigmoid sinus.

The Exenteration of the Mastoid Cells.—This is most easily and safely accomplished by means of the bone curette and rongeur forceps. All cells should be opened but not entirely obliterated, as the mucous membrane is desirable to the rapid healing of the wound. Before the operation is completed all the cellular structure of the tip and zygoma should be removed.

Exposure of the Antrum.—Careful exploration with a curved dental canal probe should be made into every small opening presented on the walls of the bony cavity. If one is found extending forward toward the middle ear, it should be cautiously followed. If it opens into the middle ear, it is probably the antrum. If it does not, it is probably a mastoid cell, and the operation should be directed still farther in search of the antrum. Unless certain of the landmarks, it is well to go very slowly, as there is a liability of inflicting injury to the facial nerve or horizontal semicircular canal. Having located the antrum, it should be freed of pus and granulations. Frequently the aditus is also found to be filled with granulation tissue which should be removed. In curetting the aditus care must be taken to not injure the

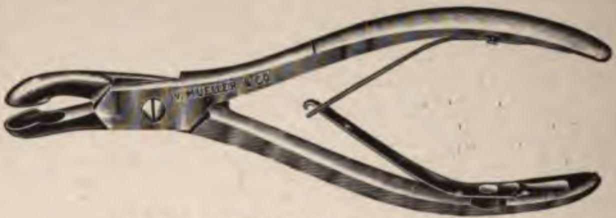

FIG. 62.—Luer-Whiting's mastoid rongeur.

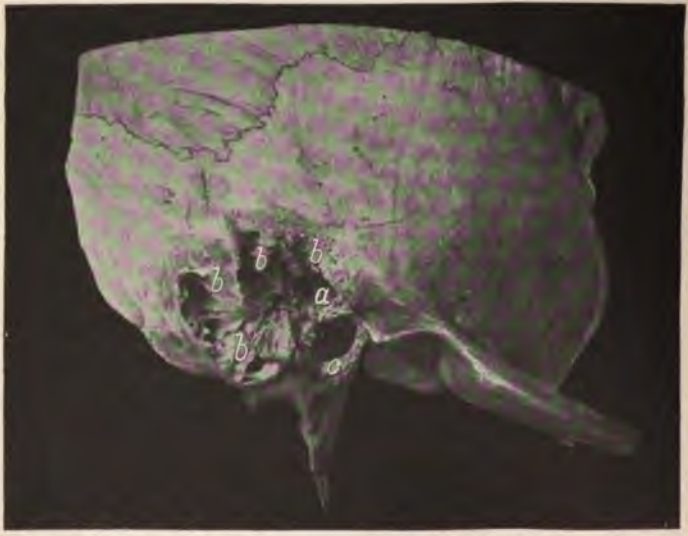

FIG. 63.—Section showing the antrum situated just above and a little behind the external auditory canal and communicating with the mastoid cells. (Bacon.)

facial nerve as it passes beneath the floor of the aditus or the horizontal semicircular canal which is situated just behind the posterior wall of the aditus.

In many cases these steps will be greatly modified by the presence of carious and necrotic bone. Where the bone is softened the operator should follow the track of carious bone to any part or in any direction. But the location of the antrum and the facial nerve should not be lost sight of. The operation should aim at a complete exposure of the antrum no matter where the carious bone may be found.

Chisels and gouges varying in width from three-sixteenths to five-eighths of an inch, the dental engine and burrs of suitable size, or the Russian perforator may

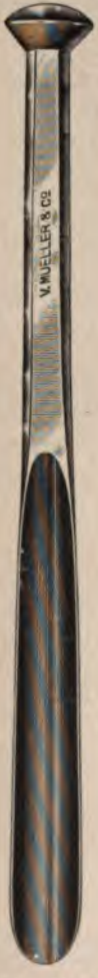

FIG. 64.—Alexander's mastoid gouge.

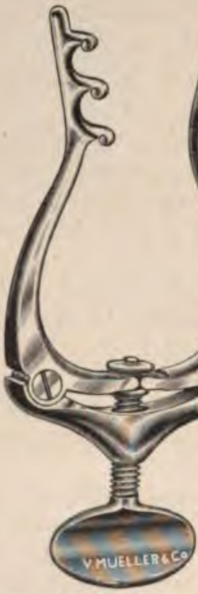

FIG. 65.—Jansen's mastoid retractor.

be used to open the antrum. The gouge is preferred by the writer.

Closure of the Wound.—Before closing the wound all the loose particles of bone and débris in the cavity should be removed either by carefully picking them out or by irrigating with warm, sterile normal salt solution.

The edges of the posterior incision (if made) are brought together and sutured. The upper half of the postauricular incision is also sutured. A narrow wick of plain or iodoform gauze should be placed in the bony wound with one end of the strip in the antrum and the other end one-half inch beyond the lower portion of incision. A similar strip of gauze should be placed in the external auditory canal. The whole ear is then covered with loosely folded gauze and a bandage applied.

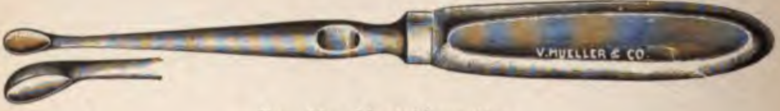
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FIG. 66.—Mastoid curettes.

The blood-clot dressing, as advocated by Blake, Reik, and others, consists in permitting the mastoid wound to fill with blood and then suturing the entire wound with silkworm gut, with the exception of the lower end, which is left open for the serous outflow.

The advantages claimed for this dressing are:

1. The blood clot is bactericidal.
2. The blood clot provides a reticulum for a new growth of bone.
3. When successful (as a large proportion of cases operated upon by this method have proven to be) a long postoperative treatment is avoided.

If this method of dressing is used, free drainage through the membrana tympani should be established by enlarging any existing perforations. The postauricular incision is made about one-half inch behind the attachment of the auricle instead of one-quarter inch.

Postoperative Treatment.—In uncomplicated cases running a normal course the first dressing may be left undisturbed for four or five days. They should be changed daily thereafter. The writer prefers to clean the wound and auditory canal with dry sterile cotton. If irrigations are used, the wound and canal should be wiped dry with sterile cotton. The stitches may be removed in from five to ten days, but sooner if there is any sign of pus retention.

Complete recovery in uncomplicated cases usually occurs in from two to six weeks.

Chronic Suppurative Otitis Media.—**Etiology.**—Chronic suppurations within the middle-ear cavity are almost invariably due to one or more attacks of acute middle-ear disease. The probabilities of an acute otitis becoming chronic are, of course, greatly enhanced by the presence of diseased adenoids and tonsils.

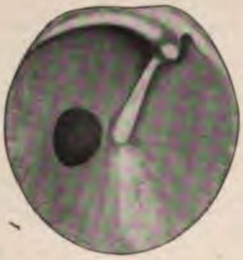

FIG. 67.—The most frequent location of a perforation in the tympanic membrane.

Pathology.—In the simpler forms of chronic suppurative otitis the mucous membrane alone may be involved. The mucosa becomes irritated and softened and soon ulcerated. Granulating surfaces may be formed even without any involvement of the bone.

In the more severe or surgical type of the disease the mucous membrane has been completely destroyed in localized areas and the underlying bone becomes carious or even necrotic with the formation of a sequestrum. The ossicles, the attic wall, or the tegmen tympani may become involved and their complete destruction may occur. This destructive process is nearly always accompanied by a partial new formation of a dense, hard bone (osteosclerosis). The cells of the mastoid may become obliterated by the reformation of the ivory-like bone.

The membrana tympani is perforated in all cases of a suppurative otitis media. The perforation may be a small pin-point opening or may embrace the major portion of the membrana tensa. The perforation is in the vast majority of cases central; however, certain long-continued suppurations show marginal perforations. Marginal perforations, as a rule, denote more or less destructive changes of the bone.

Granulating polyps may arise from portions of the denuded and carious bone and partially or completely fill the middle-ear cavity, even extending through the perforation into the external auditory canal.

As the mucosa of the middle ear is destroyed, the epidermis of the external auditory canal sometimes extends into the middle ear and replaces the mucous membrane. Nature occasionally effects a cure of the simple form of otitis in this manner; however, if the bone is necrosed, the long-continued otorrhea causes a softening and exfoliation of the superficial layers of the epidermis, with the formation of a mass of epithelial cells arranged in concentric layers and mixed with pus and cholesterin crystals. This ball or mass is called a *cholesteatoma* and when present is indicative of increased danger to the patient not only on account of the associated osseous destruction usually present with a cholesteatoma, but on account of the increased bony necrosis and absorption due to the infected, growing mass itself.

Symptoms.—The symptoms are those of a long-continued, constant or intermittent otorrhea. In neglected cases the discharge has a foul odor, although with proper treatment the odor will greatly diminish. The character of the discharge varies from a mucopurulent to a purulent. Blood cells may be found in the discharge. Bacteriological examination of the discharge as it appears through the perforated ear drum from the middle ear usually reveals a mixed infection.

Deafness of varying degree is almost always complained of in long-continued aural suppurations. The functional examination of the ear usually shows this deafness to be characteristic of the sound-conducting apparatus. This may be the result of the partial or complete destruction of the tympanic membrane, malleus and incus, or from adhesions binding and immobilizing the foot plate of the stapes in the oval window. The occurrence of the first event results in less impairment than the latter. A straight middle-ear deafness of the types just mentioned, where the labyrinth is not involved, would show a higher low-tone limit for the affected ear, a decreased air conduction and a normal or increased bone conduction. The Weber would lateralize to the affected or more affected ear. However, degeneration of the organ of Corti at the base of the cochlea frequently accompanies chronic middle-ear suppuration. In such an event there would be some impairment in the ability to hear the high tones. The bone conduction would be decreased as well as the air conduction.

Treatment.—The local treatment of chronic suppurative otitis media will effect a cure in most cases of the simple form, that is, in those cases in which the bone is not involved and in a few cases in which the bony structure of the ear is diseased. In the vast majority of the latter cases, however, operative interference is necessary to effect a cure.

There are two main objects in the local treatment (*a*) the removal of the secretions and the pathological formations such as polyps, cholesteatoma, etc.; (*b*) the maintenance as nearly as possible of a dry and sterile middle-ear cavity.

The first object, namely, the removal of the secretions, may be accomplished by irrigating two to four times a day with a warm saturated solution of boric acid or with any other solution desired, such as normal salt, permanganate of potash (pale violet color), formalin (5 drops of the 40 per cent. solution to the pint of water), 1 to 5000 bichloride solution, etc. The use of hydrogen peroxide is to be condemned as the infection may be forced from the middle-ear cavity into the mastoid or through dehiscences or breaks in the bony walls of the tympanic cavity into the surrounding structures, as described in a previous section. The writer prefers, where possible, to remove the secretions with sterile cotton mounted on small ear applicators. If the secretion is thick and tenacious, it may be necessary to inflate the ear by the Politzer or catheter method to force the secretion from the middle-ear cavity into the external canal. The discharge can sometimes be aspirated from the tympanic cavity through the perforation by means of the Siegle speculum or by means of a motor suction apparatus.

Following the cleansing of the ear it is the writer's custom to instil a warm alcoholic solution in the ear, beginning with a 50 per cent. solution and gradually increasing the strength to the 95 per cent. The alcohol serves two purposes: (1) it is antiseptic and (2) due to its hygroscopic properties it withdraws the excess water from the tissues and on evaporation leaves a dry field.

A narrow gauze wick placed along the floor of the external auditory canal so that the distal end of the wick is in contact with the perforation of the drum membrane will greatly facilitate the removal of the discharge as it forms. This wick should be removed as frequently as it becomes moist.

As the discharge becomes less in amount the insufflation of a small quantity of finely powdered boric acid will, in many cases, cause the discharge to stop. The powder should not be used in such quantity as to cause a blocking of the secretions. Exuberant granulations are best controlled by a 2 to 25 per cent. solution of silver nitrate or by the fused silver nitrate bead.

Ossiculectomy.—The malleus and incus are often more or less denuded of mucous membrane and periosteum; or they obstruct the flow of pus from the attic and antrum. When either of these conditions exist their removal is sometimes followed by a cure of the suppurative process. The handle of the malleus should be dissected from the drum-head and inflammatory fibrous tissue in which it may be imbedded. The ligaments attaching it to the attic walls should also be severed.

The whole drum-head may be removed by extending the incision around its periphery, thereby affording a clear view of the middle ear. The ligamentous attachments should be severed with suitable curved knives. The handle of the malleus should be seized near the head of the ossicle with a pair of small forceps or ring curette and pulled downward and outward with a rocking motion. The stapes should not be disturbed. The "floor" of the attic being thus removed, the flow of pus from the attic into the middle ear is unobstructed.

Aural Polypus.—Varieties.—They may be true myxomata, fibromata, angiomata, or edematous granulation tissue. They are usually the latter or of the fibromatous type, and are usually significant of caries and necrosis in the attic or antrum. They obstruct the drainage of the mastoid cells and antrum into the middle ear, and by pressure cause absorption of contiguous bony tissue. They may also hasten the extension of infection through necrotic areas in the attic and antrum.

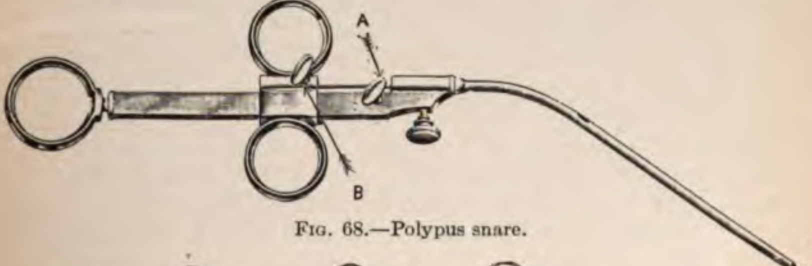

Fig. 68.—Polypus snare.

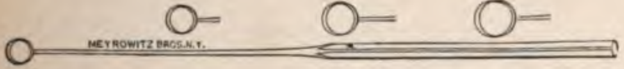

Fig. 69.—Sharp curettes.

Treatment.—They should be removed either with a small wire snare, curettes (Figs. 68 and 69), caustics, the galvanocautery, or by the instillation of 95 per cent. alcohol.

When the polypi are pedunculated and protrude into the external auditory meatus, they are best removed with a small ear snare.

If they are large, the galvanocautery applied carefully causes them to slough and shrink. Chromic acid crystals fused on the end of a probe and applied to the growths is a useful method of treatment.

When the granulations are small and succulent, alcohol instilled into the middle ear for ten to fifteen minutes every second day causes them to shrivel, and checks their growth. This is a useful adjunct to the other methods of treatment.

It should be borne in mind that polypi or granulations usually grow from the upper part of the tympanum, probably the roof of the attic, and that the bone from which it grows is probably softened or perforated by caries in which case the mastoid operation will probably be necessary to effect a cure.

Chronic Mastoiditis.—**Etiology.**—Chronic mastoiditis is, like the acute form, secondary to a preceding acute or chronic otitis media.

Pathology.—The mastoid process may contain circumscribed areas of carious and necrotic bone, or in far-advanced cases the entire process may be destroyed until the cortex is perforated, resulting in a fistula. The carious process frequently starts in the epitympanic space and in the antrum. The malleus and incus are often involved, the stapes rarely. Extension may take place in the various directions as listed under the complications of acute mastoiditis; *cholesteatoma* frequently occurs in the mastoid as a result of an extension backward of a similar process in the tympanic cavity.

The reformation of a dense, hard bone sometimes follows the destructive process. When the formation of this ivory-like bone is extensive, all the mastoid cells are obliterated and the antrum is much smaller.

Symptoms.—In most cases of chronic mastoiditis no other symptoms are present than those associated with a chronic suppurative otitis media.

Pain is absent unless drainage is interfered with to such an extent that pressure within the antrum and mastoid results.

The temperature in an uncomplicated chronic mastoiditis is normal.

Sudden deafness with dizziness and nystagmus, occurring in a previously symptomless case is very suggestive of an extension to the labyrinth or cerebellum.

Sudden cessation of the aural discharge occurring in an ear that has discharged continuously for a long time is significant of a new outlet, that is, the pus may extend to the cranial cavity or the mastoid cortex may be perforated and a subperiosteal abscess be formed. The pus may extend downward into the tissues of the neck, forming a Bezold's abscess.

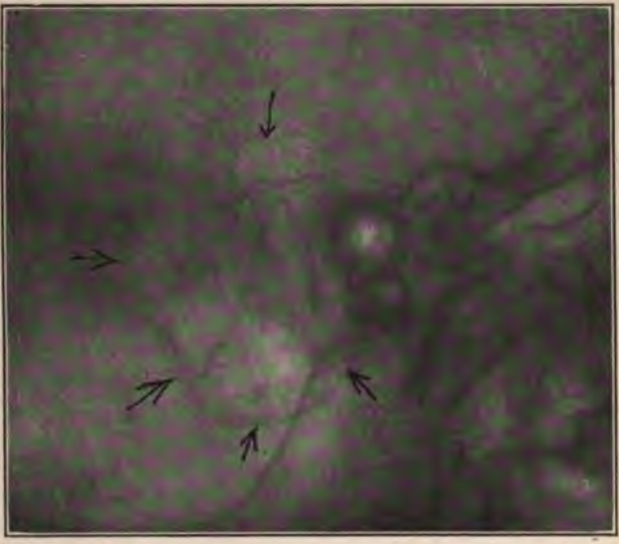

FIG. 70.—A normal mastoid as revealed by a skiagraph.

Due to the fact that the facial nerve extends a portion of its way through the middle ear and mastoid cavity facial paralysis is not an infrequent event. This paralysis may be the result of pressure or of necrosis of its outer hard shell or canal with a direct extension of the infection to the nerve.

Diagnosis.—In nearly every case of chronic suppurative otitis media the mastoid is undoubtedly more or less involved, but the extent of the involvement is not always easy to determine. The presence of aural polyps, cholesteatoma, necrosed ossicles, or postaural fistula all point to serious involvement.

The x-rays are of value in comparing the two mastoids and in determining the extent of the diseased process.

Treatment.—The local treatment is the same as that given for chronic suppurative otitis media. After a thorough trial with local measures resort to surgical procedures will be necessary to effect a cure.

Indications for the Radical Mastoid Operation:

1. Persistent ear discharge with evidence of toxemia.
2. Persistent ear discharge with pain over the mastoid, within the ear, or radiating over the side of the head.
3. Caries or necrosis in the attic or mastoid.
4. Postauricular fistula, or fistula opening into the roof, or postsuperior wall of the external auditory canal.
5. Acute exacerbations occurring during a chronic mastoiditis.
6. Cholesteatoma in attic or mastoid antrum.
7. Facial paralysis due to mastoiditis.
8. Intracranial complications.

The Radical Mastoid Operation. — **Preparation of the Patient.**—The same method of preparing the patient is used as has been described for the simple operation and will not be repeated.

The Incision.—The postauricular incision is made as in the simple mastoid operation except the middle of the incision should extend one-half to three-fourths of an inch posterior to the attachment of the auricle. This is done to prevent an excessive depression over the mastoid as might occur if the line of sutures was over the mastoid cavity.

The Removal of the Cortex and the Exenteration of the Mastoid Cells.—This is accomplished as has been described for the simple mastoid operation.

Exploration through the Exposed Antrum.—After having removed the outer wall of the antrum the cavity should be thoroughly explored by inspection and with a small probe for caries and necrosis. The presence of granulation tissue at any point is presumptive evidence of denuded bone. The granulations should be carefully removed and the base from which they spring probed for soft or roughened bone. This, if found is best removed with a sharp spoon curette.

Extreme care is necessary in working in the region of the facial nerve, which lies just under the bridge of bone left between the meatus and the operative opening made into

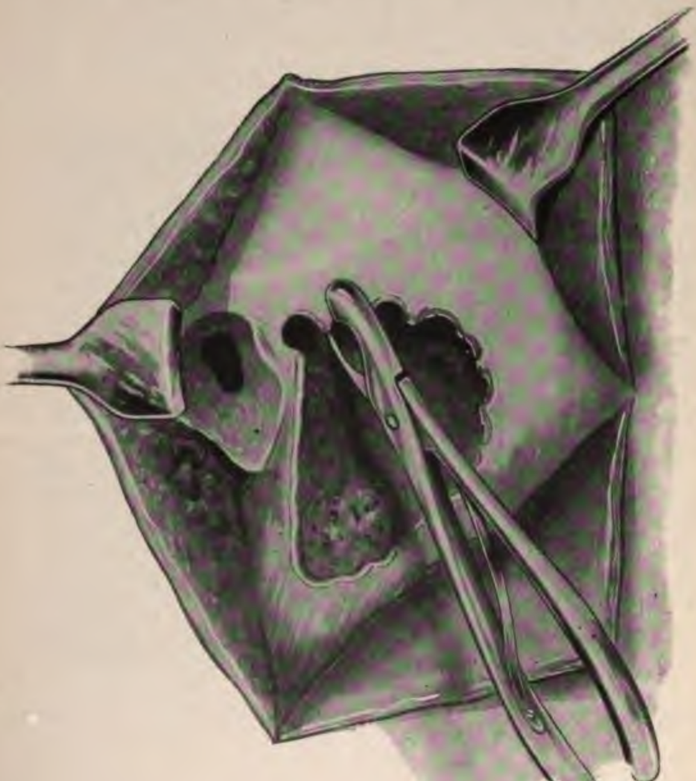

FIG. 71.—The completion of the mastoid cortex with the rongeur forceps. The cells may also be removed with the same instrument.

the antrum. It may, however, be on the inner floor of the antrum near the bridge of bone, especially that portion of the bridge below (erect position) the aditus. The aditus is the opening connecting the antrum with the attic. Carious bone in this region should be carefully curetted, the muscles of the face being watched for twitching movements.

The *middle-ear cavity* is next submitted to inspection and exploration with the probe. The ossicles and tympanic walls should be tested for bare bone and adhesive bands. If the walls of the tympanum are carious, they should be curetted. This cannot be done thoroughly with the bony wall standing between the antrum and the external meatus. Its removal is the next step in the operation.

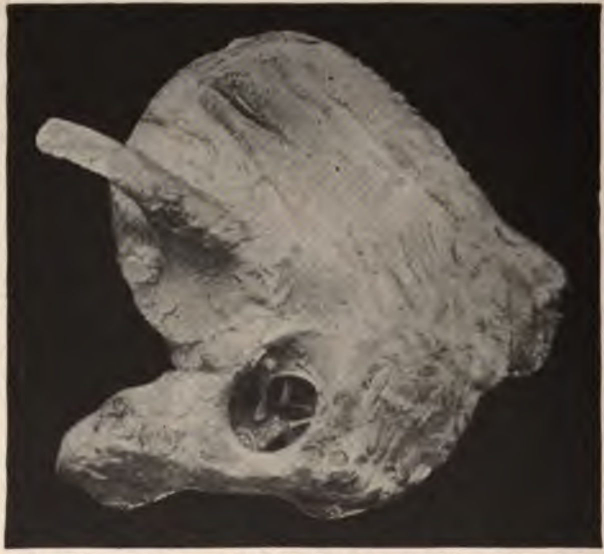

Fig. 72.—Drum-head almost entirely destroyed. Rim of membrane left in the upper portion. Ossicles bound down by adhesions. Long handle of malleus attached to inner wall of middle ear. (Bacon.)

The Removal of the Posterior Canal Wall.—This is best accomplished with gouges or the Jansen chisel. Rongeur bone forceps may be used to advantage in the superficial portion, but are not adapted to removal of the deeper portion on account of the proximity to the facial nerve and horizontal semicircular canal. In the deeper portion only the *upper part* (erect position) of the wall should be removed. The *lower portion* is usually over the facial nerve. If this precaution is observed, the bugbear of the operation is avoided, namely, the injury of the facial nerve. If chisel or gouges are used, they should be very sharp, to enable the operator to remove the bone in thin shavings. The posterior canal

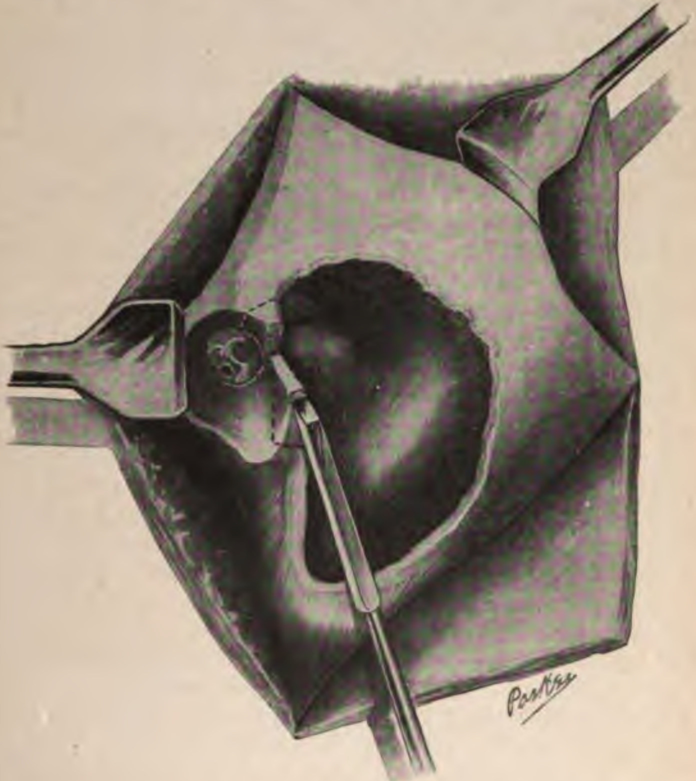

Fig. 73.—The removal of the posterior wall of the external auditory meatus down to the annulus tympanicus in the radical mastoid operation. Dotted lines indicate the amount to be removed.

wall should be entirely removed along the line of its attachment to the roof. The removal of the bridge of bone formed by the inner margin of the posterior superior canal wall (Fig. 74) is accomplished by means of a small rongeur, chisel or curette.

It is important that the small plate of bone forming the roof of the meatus be removed. This may be very satisfactorily done with a small, sharp curette. When this plate of bone is removed, the roof of the vault and the superior wall of the meatus is in a continuous line.

The Removal of Morbid Material from the Middle Ear and Mastoid Cells.—The middle-ear cavity being fully exposed to view, the *malleus* and *incus* should be carefully removed. The *stapes* should not be disturbed, as its removal would disturb the intralabyrinthine pressure or may create an avenue of entrance to the internal ear.

The *facial nerve* and semicircular canals are surrounded by dense white bone, which upon careful dissection with a burr or chisel will be exposed before the vital contents are injured. Hence the necessity of exercising extreme caution at this stage of the operation. The location of the nerve is sometimes apparent on the inner wall of the antrum, near the *aditus*, as a rounded ridge, slightly elevated above the surface.

The removal of the morbid material should be most thoroughly done, in order to prevent the continuance of the suppurative and carious processes subsequent to the operation. The most painstaking search for carious, necrotic, and granulation material should be made in every case.

All morbid material having been removed from the middle ear the Eustachian orifice should be very carefully curetted. This may be done with a small ring curette, remembering the proximity of the carotid artery.

How to Avoid Injury to the Lateral Sinus, Facial Nerve, Horizontal Semicircular Canal, and Fenestra Ovalis.—A line drawn from the parieto-squamo-mastoid junction to the tip of the mastoid indicates the position of the *sigmoid sinus*. The knee of the sigmoid sinus often extends much nearer the external meatus than this line indicates. If, however, in opening the antrum the chiselling or drilling is confined to the limits of the suprameatal triangle and directed parallel with the postsuperior wall of the canal, the lateral sinus will not often be injured. Proximity to the sinus is indicated by the presence of dense white bone. If the chiselling is carefully and cautiously done, the operator will know by this sign that he is very near it. In size the sigmoid may be compared to the size of the index finger.

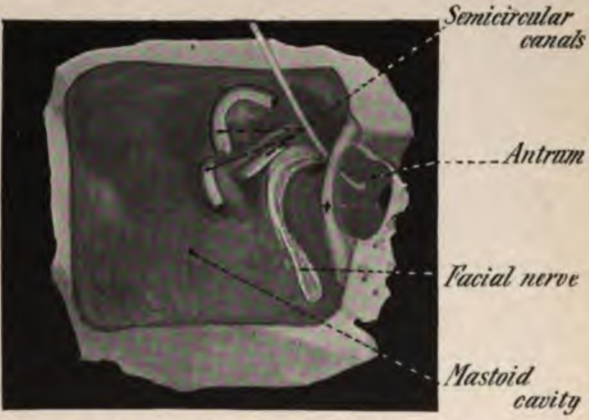

Fig. 74.—A dissection showing the relation of the facial nerve to the semicircular canals and to the antrum. (Note the bridge of bone (*x*) left from the posterior canal wall after its removal down to the annulus tympanicus). (Author's specimen.)

A knowledge of the location of the facial nerve and horizontal semicircular canal, together with the surrounding investment of dense white bony tissue, will enable the operator to avoid injuring them. Only the upper portion (erect position) of the wall between the antrum and the middle ear should be removed, as the *facial nerve* is easily injured beneath the lower part. The *horizontal semicircular* canal passes near the curve of the facial nerve, on a little deeper plane, and is therefore not so liable to injury. The facial nerve also passes beneath the superior portion of the wall between the meatus and antrum, but as it passes beneath the inner wall of the aditus is not so liable to injury during the operation.

The *oval window* need not be injured if the inner wall of the tympanum is respected during curettement of the middle ear.

The Plastic Meatal Flap.—In order that the membrano-cartilaginous canal may conform to the mastoid cavity and that a rapid epidermization of the bony wound may result some form of a meatal flap is utilized. The Ballance, the Pause, and the Körner flaps are probably the most popular ones.

The Balance incision resembles a shepherd's crook. The blades of an artery forceps are introduced into the meatus with the tips, at the inner end of the meatal tube, spread so that the tube is upon a slight tension. The Balance incision is then made through the posterior wall of the canal so that the curved portion is made in the concha.

The Körner flap is made with two parallel incisions, beginning at' the inner or tympanic end of the tube and extending into the concha. The result is a tongue-shaped flap with an attachment only at the concha.

The Pause flaps are made by making a T-shaped incision. The first incision is made in the centre of the posterior wall and the second incision is made at right angles to the first.

The excess cartilage and tissue should be dissected away from the skin flaps and the flaps then sutured in the most favorable place for the rapid epidermization of the bony cavity.

The Siebenmann-Neumann flap is a Y-shaped incision with the arms of the Y extending a short distance into the concha.

Closure of the Wound.—Having removed all morbid material from the middle ear, antrum, and mastoid cells, and having established conditions favorable for the free drainage of the same, the external wound should be partially or completely closed. A gauze wick should be inserted through the meatal opening in the concha into the mastoid cavity. Many operators prefer to use a loose packing. If a packing is used, it should be applied without great pressure. Several layers of loosely folded gauze is placed over the ear and mastoid process, and a suitable bandage applied to hold it in position.

Postoperative Treatment.—The first dressing should not be removed before three to five days, unless there are positive signs of local inflammatory reaction. The sutures may be removed in five to seven days. Subsequent dressings should be applied every day. At each dressing the middle ear and mastoid cavity should be mopped dry with sterile cotton, mounted on ear applicators. A fresh wick should be inserted and the gauze dressing reapplied.

The Results of the Operation.—The objects of the operation are: (*a*) To avert impending danger from the progress of caries and necrosis in the course of secondary acute and chronic mastoid disease. In secondary acute mastoiditis the bony tissues undergo very rapid degeneration; while in the chronic form the process is a much slower one, oftentimes giving rise to no marked symptoms. (*b*) To establish permanent free drainage of the mastoid cells, antrum, and middle ear.

Chronic Non-suppurative Otitis Media.—Synonyms.—Chronic Middle-ear Catarrh; Dry Middle-ear Catarrh; Hyperplastic Middle-ear Catarrh; Adhesive Catarrh of the Middle Ear.

Etiology.—This disease is undoubtedly due, in most cases in the adult, to repeated attacks of untreated acute tubal catarrh and acute catarrhal otitis media, hence the factors which produce the acute catarrhal conditions will produce the chronic non-suppurative otitis media.

The presence of neglected adenoids in children is probably one of the most common agents in the production of this disease. Frequent attacks of acute rhinitis and obstructing nasopharyngeal lesions are also etiologic factors of importance. Other causes are heredity, age, sex, climate, and occupation, as discussed in a previous section. Constitutional diseases such as anemia, diabetes etc., and general diseases such as syphilis, tuberculosis, typhoid, etc., act as predisposing causes.

Pathology.—The pathological process may be divided into two stages.

1. The *exudative stage* which may be associated with a subacute or chronic tubal or tubotympanic congestion.

The swollen or thickened mucous membrane of the Eustachian tube and tympanic cavity results in a rarefaction of the air within the middle ear and a retraction of the tympanic membrane. Edema and the exudate of fluid into the tympanic cavity follow. When this condition persists for some time a true tissue hypertrophy of the mucous membrane takes place with a more or less permanent diminishing of the lumen of the Eustachian canal. The pathologic process may progress no further but persist as a chronic catarrhal otitis media.

2. The *hyperplastic stage* usually follows the preceding but has been noted to occur independently of the exudative stage. Closely associated with the exudative process is the formation of new connective tissue which forms bands and adhesions binding the ossicles together and to the adjacent walls.

The foot plate of the stapes may be immobilized within the oval window, adding greatly to the impairment of hearing. The lining mucous membrane of the tympanum becomes thick and dry. Lime salts may be deposited on the drum membrane or within the ossicular articulation, thereby increasing the immobility.

Symptoms.—The chief symptoms complained of by the patients are gradually increasing deafness and head noises. The deafness may not be noticed by the patient if the disease is unilateral until the functional examination shows a marked impairment of hearing for the affected ear. In the hyperplastic form of the disease the patients frequently hear better in the presence of noises than in a quiet room (paracusis Willisii).

The tinnitus aurium varies greatly in intensity in different cases and even in each individual. When tired or worried, the noise becomes more pronounced. The tinnitus is continuous in well-advanced cases.

Pain is rarely present.

The drum membrane is thickened, lustreless and opaque; white areas of calcareous deposit can sometimes be seen. The drum membrane is retracted. The handle of the malleus is foreshortened, the short process is more prominent than normal, the cone of light is misplaced, broken, or absent. Circumscribed retractions of the drum membrane may be present from adhesions and cicatricial bands. Atrophic areas can sometimes be seen in the drum membrane.

The functional examination reveals a reduced hearing distance for the voice, whisper, and watch. In cases uncomplicated by labyrinthine deafness the low limit is raised and the high limit is normal or only slightly lowered. Bone conduction is increased and air conduction decreased. The Weber is heard better in the worst ear.

Prognosis.—The prognosis in the majority of cases is unfavorable as to the relief of the tinnitus aurium and the restoration of function. This is especially true of those cases with a permeable Eustachian tube. If the tube is closed, improvement may be expected with the restoration of its normal patency. The disease can often be arrested upon correcting a coexistent nasal or nasopharyngeal disease.

Treatment.—To relieve the tinnitus and to restore the function it is necessary that the nose and nasopharynx be in the best possible condition. This may necessitate the removal of diseased adenoids and tonsils or correction of the various abnormalities of the nose.

The next step is the restoration of the normal patency of the Eustachian tube. This is best accomplished by means of catheterization. If the patient notices any relief, the inflation should be given every two or three days for a week or so and then at longer intervals until patency is assured.

The inflation of medicated vapors such as menthol, camphor, eucalyptol, etc., may be used instead of air.

Frequently inflations cannot be satisfactorily carried out, due to constriction of the Eustachian tube. In this event it is necessary to enlarge the constrictions by means of the Eustachian bougie. If mucus is present in the nose or nasopharynx this should be removed by wiping with dry cotton or by spraying.

Method of Introducing the Bougie.—(1) Insert a catheter with a lumen large enough to allow the bougie to pass freely. (2) Inflate the tympanum, and with the diagnostic tube determine if possible whether or not the catheter is within the Eustachian orifice. (3) Introduce the bougie, previously dipped in sterile vaseline, into the catheter until it reaches the Eustachian tube. It should then be carefully and gently pushed into the tube and beyond the stricture, and left there for several minutes. Extreme care must be observed to avoid forcing the catheter through the mucous membrane. It is well to tie a thread around the bougie at a point marking the length of the catheter, so as to know when the tip of the bougie is at the Eustachian orifice. The bougie may be introduced an inch or an inch and one-eighth without danger of injuring the contents of the tympanum. Bougies of graduated sizes may be introduced in succession or at successive treatments. The treatments should not be repeated oftener than two or three times a week, but should be continued until the stricture shows no signs of contraction. The bougies may be made of whale-bone or other flexible material. If desired bougies made of catgut and inpregnated with silver nitrate may be used.

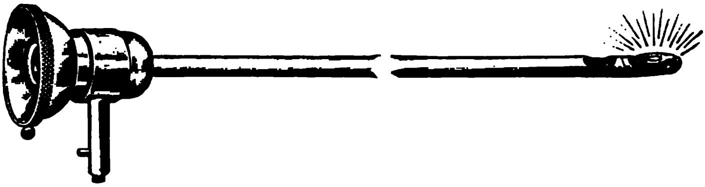

FIG. 75.—Holmes' nasopharyngoscope.

In the exudative form of the chronic non-suppurative otitis media the exudate may be removed by the various methods given for the treatment of acute catarrhal otitis media. Massage of the tympanic membrane and the ossicular chain by means of Delstanche's masseur and Siegle's otoscope (Fig. 76) has been tried for a number of years with but little success. The object of the alternate rarefaction and condensation of the air, produced by the two instruments mentioned, is to stretch or break the adhesions producing the ankylosis.

The bromides are frequently given internally for the relief of the advanced tinnitus aurium, and in extremely nervous patients chloral hydrate or morphine may be used to secure a night's rest. Potassium iodide is widely used and should be prescribed in all cases where syphilis is suspected. Pilocarpine hydrochlorate, $\frac{1}{6}$ of a grain given hypodermically at bedtime, has given good results in many cases where the intralabyrinthine pressure is increased, due either to an excessive exudate or to pressure from an ankylosed foot plate of the stapes in the oval window.

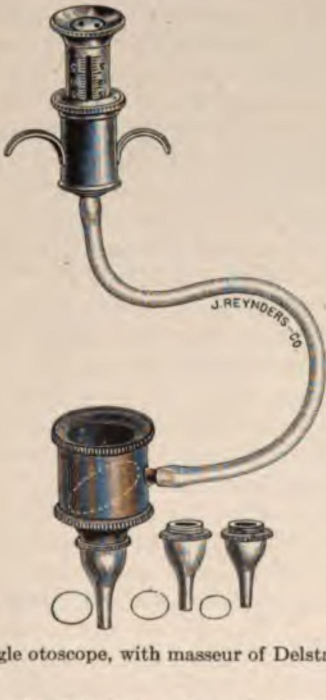

Fig. 76.—Siegle otoscope, with masseur of Delstanche attached.

Various surgical procedures have been proposed for the relief of this disease but the results have not been brilliant, indeed many cases have been made worse. Some of these measures are:

1. Multiple incisions of the tympanic membrane.
2. Section of the posterior fold (plicotomy) where marked

retraction of the drum membrane exists. This is sometimes done in cases of great deafness with subjective noises and pronounced retraction of the handle of the malleus. The section is made immediately behind the short process, or midway between the process and the peripheral end of the fold, cutting from above downward. The handle of the malleus is thus allowed greater motion, the subjective noises are diminished, and there is improvement of hearing lasting for a few weeks or months.

3. Partial excision of the drum membrane where it is greatly thickened or calcified.

4. Tenotomy of the tensor tympani muscle.

5. Removal of the malleus and incus where marked ankylosis is present. If there is ankylosis of the malleus and incus, or firm adhesive bands interfering with their mobility, the bones may be extracted through an incision in the drumhead. This operation was once quite popular, as it promised great relief in this type of deafness. A larger experience has demonstrated that the beneficial results in most instances are of temporary duration.

Adhesions, Thickening, and Retractions following Chronic Exudative Inflammation of the Middle Ear.—Fibrous Bands.—Fibrous bands are left as a result of chronic inflammation.

Effect on Membranes.—They may bind the membrane to the inner wall of the tympanic cavity or to the incus and stapes. When the drum-head thus becomes adherent, exhausting the air in the external auditory meatus with the Delstanche masseur and the Siegle otoscope (Fig. 76) does not cause an outward movement of the entire drum-head. The adherent portions remain fixed, while the rest of the membrane moves outward.

The bands may also extend from the ossicles to the inner wall of the tympanum. All these conditions are attended by more or less deafness and tinnitus.

Effect on Tensor Tympani.—The fibrous formations and the subsequent retraction sometimes cause the tendon of the tensor tympani muscle to be shortened. The focussing and conduction functions of the drum-head and ossicles are thus impaired.

Effect on Cavity of the Tympanum.—This is often very much diminished in size by the chronic hyperemia, hypertrophy, and hyperplasia of the mucous membrane.

Otosclerosis.—Otosclerosis is a chronic disease characterized by a spongification or porosis of the bony capsule of the labyrinth, by loss of movement of the stapes, due to a bony ankylosis, by a normal tympanic membrane, by a permeable Eustachian tube, and by a typical sound-conduction deafness.

Etiology.—The factors concerned in the production of otosclerosis are not clearly understood. Many theories have been presented but none of them proven. Age exerts a positive influence upon the development of the disease. It usually begins between the eighteenth and fortieth years. Women are more frequently subjected to the disease than are men. Heredity is undoubtedly a factor in many cases, as numerous instances are on record giving the history of other members of the family having had the disease. Parturition aggravates the symptoms and in a few cases the beginning of the disease dates from the birth of the first child. Syphilis, rheumatic and gouty diathesis, and neurasthenia have been given by some authors as having something to do with the production of the trouble.

Pathology.—The first pathologic change consists in the resorption of the cartilage cells situated in the osseous capsule of the labyrinth or the ossicles. The cartilage cells are most common in the posterior half of the margin of the oval window. The capsule of the semicircular canals and the upper and lower walls of the cochlea also contain these cartilage cells. According to Denker, the first osseous change consists in an active proliferation of all the cellular elements within the bone. In the new-formed bone giant cells are found. Hollow spaces are formed, and cavities of erosion gradually form in the original compact bone. Associated with the absorption of the cartilage and bone is a formation of new porous bone. This new-bone formation later assumes a lamellar structure. These bony changes usually take place about the foot plate of the stapes, in the cochlea, the vestibule, and the semicircular canals. The foot plate of the stapes may become completely ankylosed in the oval window.

The alteration in the structure of the bone of the labyrinth may produce a change in the pressure of the labyrinthine fluid.

Symptoms.—The onset is insidious and usually bilateral, although one ear may be involved earlier than the other or may be more severely involved.

The tinnitus may precede the deafness, both gradually increasing until the patient has great difficulty in hearing the voice even a few inches from the ear. A very common phase of the deafness in otosclerosis is the ability to hear better in a noisy place, as when riding on a street car or railway train, than in a quiet place (paracusis Willisii).

The tinnitus aurium is present in almost every case of otosclerosis and is complained of most bitterly. Various nervous disorders may follow in susceptible individuals as a result of the distressing tinnitus. Worry, mental or physical fatigue make the noises worse.

Diagnosis.—The diagnosis is based upon a history of a gradually increasing tinnitus and deafness with a normal tympanic membrane and Eustachian tube together with the findings of the functional examination.

The functional examination of the ears where the hyperostosis or the spongifying process is confined to the region of the stapes and oval window reveals a raised low-tone limit with a normal or very slight lowering of the high-tone limit. Air conduction is decreased and bone conduction is increased. The Weber test shows a lateralization of the sound to the more affected ear. If in addition to the above, the cochlea is involved, the high limit is lowered by air conduction, the air conduction is greatly lessened and the bone conduction is not increased to the extent found in the first-mentioned condition.

Prognosis.—The prognosis as to the restoration of function and the relief of the tinnitus is very unfavorable. The deafness probably never becomes absolute. The tinnitus becomes more endurable as time goes on. The patient need have no anxiety concerning his condition other than the handicap of the deafness and the annoyance of the head noises. These patients can usually hear well over the telephone or by artificial hearing devices.

Treatment.—The local treatment of otosclerosis is useless, indeed in many cases the over-use of the catheter, and the pneumomassage appears to make the symptoms worse. There are many advocates of the restricted use of the pneumomassage. The claim is made that ankylosis of the foot plate of the stapes in the oval window is prevented or made less.

The internal administration of potassium iodide and mercury may be given, particularly if a specific origin of the disease is suspected. Small doses of phosphorus, gr. $\frac{1}{200}$, three times a day, for some months have given good results in many cases. Thyroid extract has improved some cases.

The nose and throat as well as the general health should be kept in the best possible condition. As soon as a definite diagnósis of otosclerosis is made the patient should be advised to take up the study of lip-reading. It may be explained that while lip-reading will not be absolutely necessary to carry on a conversation it will nevertheless greatly facilitate the process.

Intracranial Complications—Thrombosis of the Sigmoid Sinus.—Etiology and Complications.—Thrombosis of the sigmoid sinus may occur in the course of chronic suppurative inflammation of the mastoid cells, either from close proximity or venous communication (Fig. 77). If the thrombus disintegrates and spreads throughout the blood, septicemia or pyemia results. The particles of floating thrombus may lodge in the lungs, liver, kidneys, or other parts of the body, and give rise to septic foci of inflammation. If the lungs are the lodging place of these septic thrombi, frequently a rusty sputum and in some cases a gangrenous odor may be noticed. Purulent basic leptomeningitis may occur on the median side of the sinus, and there may be a cerebellar inflammation with abscess.

If the thrombus is aseptic, it may be absorbed and leave the sinus thickened, contracted, or obliterated.

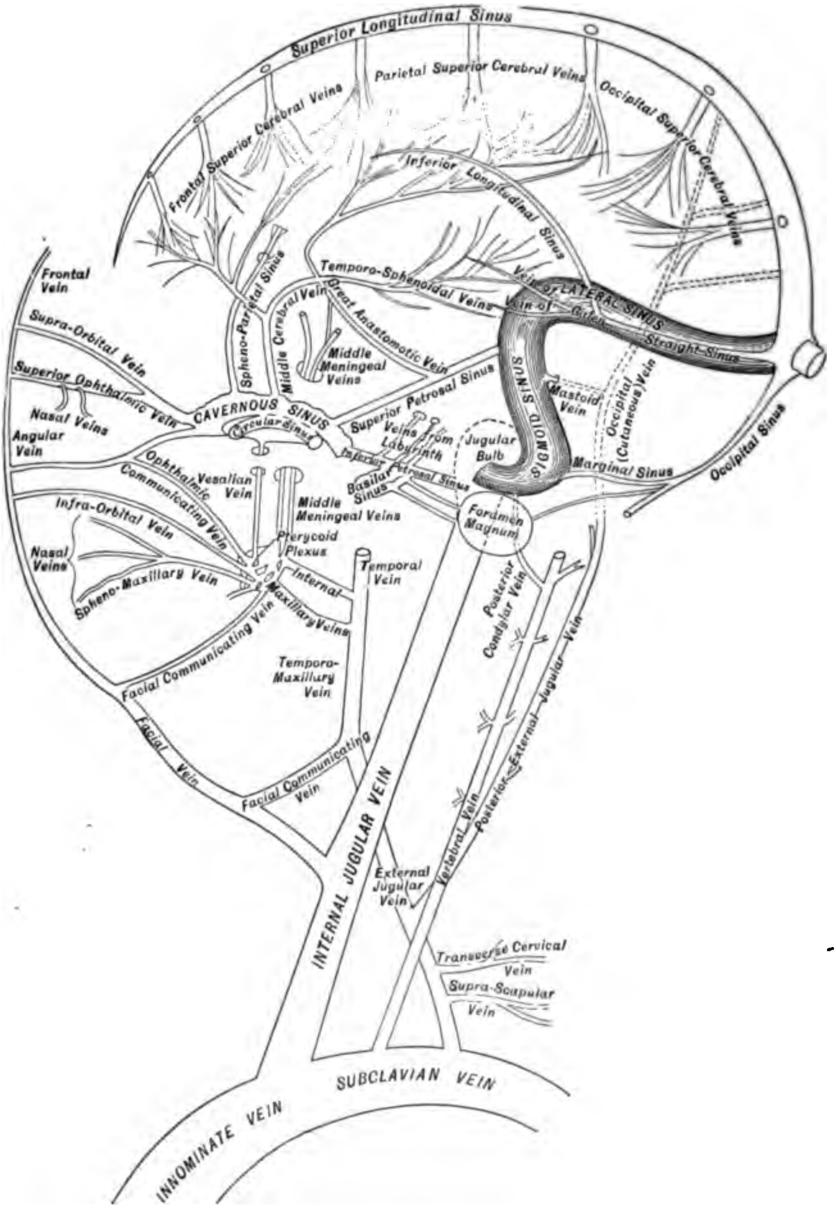

Fig. 77.—Cerebral veins and sinuses. (From Macewen.)

Symptoms.—Severe rigors at intervals of one to three days, followed by profuse perspiration, are the most characteristic symptoms. A high but *variable* temperature is present during the height of the process. A rapid and increasingly weak pulse marks the progress of the disease. The pulmonary signs already referred to, and vomiting and diarrhea, are also symptoms of importance. Giddiness is sometimes present, and the intellect remains clear in cases uncomplicated with meningitis. Pain on percussion and pressure is present on the posterior part of the mastoid. Edema due to obstruction in the emissary veins of the skull is sometimes present.

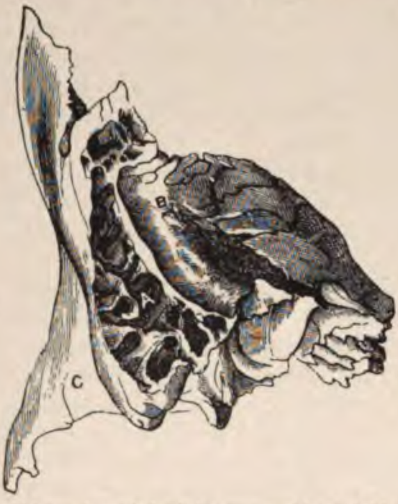

Fig. 78.—Mastoid portion of the left temporal bone laid open and viewed from behind: *A*, mastoid cells extending from the mastoid process below, upward, and inward, over the lateral sinus *B; C*, the zygoma. (Burnett.)

The blood count should be made early. A high polymorphonuclear count is very suggestive when combined with the above symptoms.

A blood culture should also be made.

Septic thrombi occurring in the other venous sinuses of the skull give rise to symptoms peculiar to their anatomical relations.

Prognosis.—It usually runs a course of two or three weeks, and unless operative interference is practised terminates fatally. Metastatic abscesses in the lungs, liver, spleen, and kidneys render the prognosis almost certainly fatal. It therefore becomes important that an early diagnosis and operation be made in order to remove the clot before it disintegrates and infects other organs of the body.

Treatment.—The mastoid operation (see page 252) should be done, the sinus exposed along its mastoid portion, and the fibrous wall of the vessel freely slit. The clot should be thoroughly removed, the wound packed with plain or iodoform gauze, and the outer pushed against the inner wall and allowed to become adherent, and thus close the sinus. Another mode of operating is to ligate the jugular vein in the neck and then remove the clot. These operations are attended by the cure of many cases which would otherwise prove fatal.

The **lateral superior petrosal,** and **cavernous sinuses** are more rarely involved. Impaired vision, exophthalmos, and edema around the eyeball are signs of cavernous sinus thrombosis.

Menigitis of Otic Origin.—A much larger proportion of the reported cases of meningitis have their origin in chronic suppurative otitis media than is generally supposed.

It is in the old chronic cases, in which the discharge has been scanty, that cranial involvement is most apt to occur. The reasons for this are given under *Pathology* (p. 245). The discharge from the ear may have been so slight as to have escaped the notice of the patient or family physician, and yet a careful inspection would show pus which has become dried and exfoliates with epithelial scales of the external meatus.

Leptomeningitis of Otic Origin.—**Definition.**—This is a serous or purulent inflammation beginning in the arachnoid and extending to the contiguous membranes and superficial portion of the brain. It is an exceedingly fatal disease, and may run a rapid course, although in some cases it extends over several weeks.

Symptoms.—The symptoms are headache, restlessness, insomnia, photophobia, retracted abdomen, contracted pupils, hypersensitiveness of the skin, full, bounding, rapid pulse, high temperature, and optic neuritis. As the disease progresses the intellect becomes dulled, the pulse slow, and the respiration rapid. Paralysis finally supervenes, and the urine, and feces are discharged involuntarily. The later signs point to a speedy termination.

Surgical Treatment.—In addition to the usual remedies, the mastoid may be opened as described and the middle cranial fossa, the lateral sinus, and neighboring parts explored and freed from necrotic and other morbid material. Irrigation and drainage sometimes avert a fatal issue. The operation seems justifiable at times, as without it a cure can hardly be hoped for.

A lumbar puncture may be done to obtain a specimen of the cerebrospinal fluid for microscopic and chemical examination as an aid to diagnosis or as a means of treatment for serous meningitis.

Hayne's operation may be done although the results have not been all that were hoped for.

Extradural Abscess.—**Etiology.**—When in the course of chronic suppurative inflammation of the attic, antrum, or mastoid cells, the bony walls become necrosed and perforated, pus sockets may form between the dura and the skull.

Treatment.—They should be thoroughly exposed and adequate drainage established. The preliminary step in the operation is the radical mastoid operation. When this operation is done, intracranial abscesses are found that would have otherwise escaped observation until more serious involvement occurred.

Cerebral and Cerebellar Abscess.—**Etiology.**—This may be a later manifestation of an extradural abscess which has eroded through the dura and penetrated the brain, or it may be due to a thrombus carried by the small cerebral arteries. Infection may also gain entrance through the perivascular sheaths. A few cases have been reported as occurring in children where infection has taken place through the petrosquamosal suture. Tubercular disintegration of the petrous portion of the temporal bone leads to tubercular abscess of the temporosphenoidal lobe, and is usually associated with perforation of the roof of the attic or antrum. This is important to remember, as in operating it is desirable to get at the seat of the trouble, remove all morbid material, and establish free drainage through the middle ear.

In mastoid operations extreme care should always be taken to examine the roof of the attic and antrum, as well as the walls of the lateral sinus, otherwise the wound will sometimes be closed and the existing brain or extradural abscess overlooked until serious and perhaps fatal brain disease has developed.

Symptoms.—Oftentimes the first sign is the cessation or partial suppression of the ear discharge. During the *stage of invasion* there may be nausea, vomiting, pain, and mental irritability. Chilly sensations may be complained of. The normal temperament is often reversed—that is, a vivacious, socially inclined person becomes morose and seclusive. During the second stage or *stage of increasing intracranial pressure* cerebration becomes dull, the pulse quite slow, and the temperature subnormal or but slightly elevated. Occasionally there may be high temperature, rapid pulse, rigors, and convulsions, as in thrombosis of the sigmoid sinus.

Aphasia in this connection is very suggestive of abscess of the temporosphenoidal lobe. In right-handed people it is a sign of abscess on the left side, while in left-handed persons it is a sign of abscess on the right side.

There may be optic neuritis in cerebellar abscess, and, rarely, in abscess of the cerebrum. In cerebral abscess the percussion note over the mastoid is sometimes high. This is due to the sclerosis which has occurred in the mastoid as a result of the chronic suppurative inflammation. This also accounts for the perforation being upward through the roof of the attic and antrum rather than backward through the mastoid process.

The reflexes of the limbs on the opposite side from the abscess are usually increased.

The characteristic symptoms of abscess in the *cerebellum* are slow pulse, low temperature, headache, nausea, vomiting, staggering gait, vertigo, nystagmus, and facial paralysis.

Many cases of diffuse or circumscribed suppurative labyrinthitis present the above symptoms; hence the diagnosis is not always easy. The establishment of free drainage is sometimes followed by relief of the symptoms of intracranial abscess. This does not necessarily prove that brain abscess did not exist, as it may drain through the perforation in the roof of the attic or antrum, after the removal of the granulations which obstructed it.

The *nystagmus* in cerebellar abscess is frequently a changing one, that is, the quick component may be directed to the diseased side (usually the case) or it may be directed to the normal side. It may change its direction and be first to one side and then to the other. This changing nystagmus is in marked contrast to the labyrinthine nystagmus, which does not change in direction when the eyes are voluntarily turned except in some cases of circumscribed labyrinthitis. Cerebellar nystagmus tends to persist. The nystagmus of labyrinthine disease tends to gradually subside after loss of function.

The Pointing Test.—Barany has shown that a person with a cerebellar abscess with focal symptoms is unable to locate an object (patient blindfolded) with the hand corresponding to the cerebellar lesion, while he is able to do so with the other hand. With vestibular nystagmus both hands deviate in the same direction, *i. e.,* in the direction of the slow component of the nystagmus.

Adiadokokinesis, first mentioned by Babinski, is the ability to execute rapidly, successive volitional movements. For example, if the patient is asked to alternately pronate and supinate the forearm upon the arm, movements of the arm corresponding to the cerebellar lesion will be slower and less regular, although the muscular force is preserved and sensibility is intact.

Sometimes the Cheyne-Stokes respiration, apathy, intolerance of light, yawning, and rigidity of the muscles of the neck and jaws are present.

Prognosis.—Without operative treatment nearly all cases end fatally. Under operative treatment about 30 to 50 per cent. recover.

Dench has collected 100 cases of cerebral abscess which were operated; of the 100 cases, 52 recovered and 48 died.

Treatment.—It is usually best to do the mastoid operation, converting the middle-ear cavity, attic, antrum, and mastoid cells (if need be) into one large cavity which can be freely inspected and explored for caries and perforation. If the temporosphenoidal lobe is the seat of the abscess, a perforation will usually be found through the roof of the attic or antrum. If possible, the abscess should be searched for through the perforation and its contents drawn off with a syringe, the needle of which is at least 1 mm. in diameter.

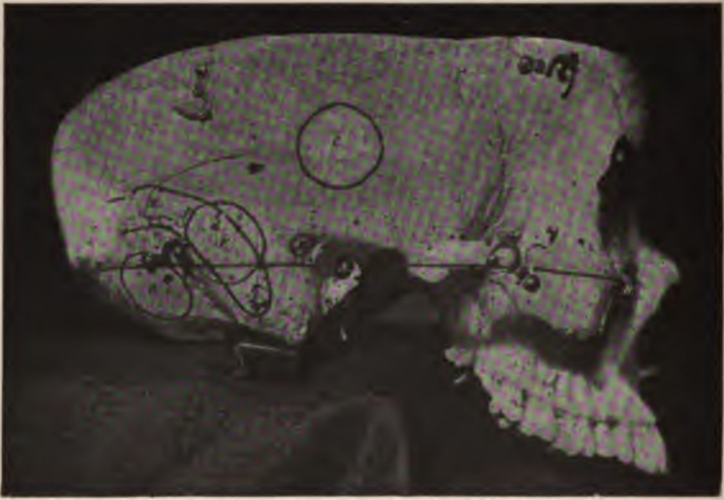

FIG. 79.—*A*, external auditory meatus; *B*, *B′*, Reid's base-line running from the lower margin of the orbit through the centre of the external auditory meatus; *C*, trephine-opening, three-quarters of an inch in diameter, to expose temporosphenoidal abscess, the centre-pin of the trephine being placed one and one-quarter inches above the centre of the external bony meatus; *D*, point at which the mastoid antrum should be opened; *E*, *E′*, position of sigmoid sinus; *F*, trephine opening, three-quarters of an inch in diameter, for exposing sigmoid sinus, the centre-pin of the trephine being placed at a point one inch behind and one-quarter of an inch above the centre of the external bony meatus; *G*, trephine opening for cerebellar abscess, the centre-pin being placed two inches behind and one-quarter of an inch below the centre of the external bony meatus.

If it cannot be reached through the point of perforation, the skull should be trephined at *C* in Fig. 79.

Through this opening the needle should be introduced and part of the contents of the cavity withdrawn, which should then be laid open with a scalpel, care being taken to avoid cutting the arteries. All sloughs should be carefully removed with a spoon curette before closing the wound. A strip of sterilized boric-acid gauze should be introduced loosely into the abscess cavity for drainage. This should be changed daily until the amount of discharge materially decreases.

When the abscess is in the *cerebellum*, the mastoid operation should also be performed, and the lateral sinus examined and freed from clots if they are present. The cerebellum should then be exposed by removing the bone around the sinus or by removing a button of bone at the point *G*, Fig. 79. The pus should be drawn off with a hypodermic syringe or the cavity freely laid open with a scalpel, and a loose gauze dressing introduced. Many operators prefer to use a glass or silver drainage tube instead of the gauze wick.

Paresis and Paralysis of the Auditory Nerve.—Angioneurotic Type.—The angioneurotic type of auditory paralysis is rare, and is characterized by sudden pallor of the face, which is immediately followed by giddiness, nausea, tinnitus, vertigo, and deafness. After a few moments the pallor and ear symptoms disappear, leaving no deafness or trace of ear disease.

Rheumatic Paralysis.—Rheumatic paralysis of the auditory nerve occurs occasionally in the course of rheumatism in other parts of the body. There is usually complete deafness, which gradually disappears.

Hysterical Paralysis.—Hysterical paralysis is characterized by an unstable and eccentric nervous system, and deafness which appears and disappears rapidly without visible signs of ear disease to account for it.

Treatment.—This will depend upon the duration of the deafness and the causes of the same. The more acute the case the more favorable is the result of the treatment. Rest in a quiet room and the use of hot foot-baths with mustard, vesicants over the mastoid, and saline purgatives often favor a rapid recovery. *Local treatment*, as inflation, etc., is contraindicated in most cases, especially when there are no signs of middle-ear disease.

THE INTERNAL EAR.

INJURIES.

Etiology.—Injuries to the internal ear may occur from *direct violence*, as gunshot wounds; and sharp instruments, etc., introduced accidentally through the external auditory canal. The instrument may penetrate the oval or round windows and allow the intralabyrinthine fluid to escape.

Injuries from *indirect violence* may result from a blow upon the skull, by which the petrous portion of the temporal bone is fractured; or from a blow upon the ear, usually in the presence of a diseased Eustachian tube in which the air is condensed in the meatus and middle ear and driven upon the oval and round windows. The terminal expansion of the auditory nerve is thus injured. A loud cannon report may also cause condensation in the middle ear, and thus injure the terminal auditory nerve endings.

Symptoms.—The symptoms of concussion of the labyrinth vary with the degree of injury, from slight stupefaction and singing noises of a few hours' or days' duration, to immediate and marked deafness with loud subjective noises, confusion of ideas, and giddiness. After a time the subjective noises improve or change to a shrill metallic noise, especially in the presence of objective noises. The hearing often remains unimproved. Bone and air conduction is much diminished or altogether wanting, according to the impairment of hearing. The vibrating tuning-fork on the vertex is heard best on the uninjured side.

Fracture of the base of the skull which involves the internal ear may result in a permanent total deafness. It is evident that in an injury of this type in which all hearing is gone that the tinnitus would be absent also.

Prognosis.—The prognosis in most cases of concussion is unfavorable so far as a return of hearing is concerned. Sometimes there is improvement after many weeks or months; while in other cases there may be progressive increase in the deafness and subjective noises.

DISEASES OF THE INTERNAL EAR.

Hyperacuteness of Hearing.—**Synonyms.**—Hyperesthesia Acoustica; Oxyecoia.

Definition.—This is a rare condition in which there is a *temporary increase* in the acuteness of hearing.

It may be *due to* intracranial irritation, local degeneration, the irritation occurring at the onset of infectious fevers, meningitis, hemicrania, and trigeminal neuralgia.

Paracusis.—This is a condition of the auditory nerve or nerve endings which is characterized by *perverted perception* of the pitch of musical sounds, sometimes amounting to five tones. Slight changes in pitch may be due to altered tension of the sound-conducting apparatus; hence care should be exercised to differentiate the two conditions.

Paracusis Loci.—Paracusis loci is the inability to determine the direction of the source of sound. Sound upon the deaf side may be thought to come from the opposite side, and may prove a source of danger.

Paracusis Diplacusis.—One ear having an altered tone perception, a sound is recognized by the two ears as a double tone, usually quite discordant in character. Both ears may be unequally out of tune, and thus give rise to the phenomenon.

Paracusis Willisii.—This is a condition frequently occurring in the course of otosclerosis, and is characterized by ability to hear better in the presence of objective noises, as in a moving railway train or on the street cars.

Subjective Noises in the Ear; Tinnitus.—The following quotation from Politzer gives in brief form the varying types of subjective noises experienced as the result of direct or reflex irritation to the auditory nerve:

"The subjective sensations of hearing are characterized by the patient in different ways—most frequently as whizzing, roaring, buzzing, seething, hissing, singing, ringing, humming, and whistling in the ear. The character of the noises is designated as either high or low by patients who are capable of judging."

Etiology and Occurrence.—The conditions of atmosphere and climate which render catarrhal diseases worse, increase the noises. They are usually worse at night and in quiet places. They may be intermittent or constant. They may occur in persons of a nervous, irritable temperament without accompanying deafness. This type usually continues throughout life.

Treatment.—The *cause* should be determined and removed or ameliorated if possible, such as existing external-, middle-, or internal-ear disease; coexisting nasal and nasopharyngeal diseases; anemia or other constitutional disorders.

There are certain *local measures* which afford relief in a considerable number of cases. The most successful are inflation of the tympanic cavity by the Politzer method; inflation by catheterization; and pneumatic massage of the external auditory meatus with the Delstanche masseur or the electric pump. The injection of medicated oils into the tympanic cavity through the catheter is also sometimes attended by relief. Artificial perforation of the drum-head affords temporary relief in cases in which there is great retraction of the drum-head from closure of the Eustachian tube. The Eustachian bougie is occasionally of service.

Hyperemia, Anemia, and Hemorrhage of the Labyrinth.—**Hyperemia.**—Hyperemia is found in acute purulent inflammation of the tympanum; in the acute exanthematous fevers; in meningitis; and in diseases of the lungs and heart. The administration of the nitrite of amyl, salicylic acid, and quinine may also cause it.

Symptoms.—The symptoms are giddiness, vertigo, staggering gait, nystagmus, tinnitus, and a feeling of fulness in the ears.

Treatment.—The treatment should be addressed to the cause.

Anemia.—It may occur in general anemia and chlorosis, after great loss of blood, and in tuberculosis. A rapid pulse with deafness, vertigo, nausea, vomiting, pronounced tinnitus, and faintness should excite suspicion of pulmonary tuberculosis.

Prognosis.—The prognosis in these cases should be guarded.

Hemorrhage.—It may *occur* from fracture of the base of the skull; severe concussions to the head or the external ear; necrosis of the petrous portion of the temporal bone; and from atheromatous changes in the walls of the bloodvessels of the labyrinth. The hyperemia attending the exanthematous fevers may also give rise to slight hemorrhages.

Increased Labyrinthine Pressure.—**Etiology.**—Catarrhal and adhesive processes in the middle ear may cause increased labyrinthine pressure by adhesive processes with contraction of the adhesive bands, whereby the foot plate of the stapes is held forcibly against the oval window. Adhesive bands and calcareous deposits sometimes interfere with the expansile properties of the membrane of the round window, and may thus increase the intralabyrinthine pressure.

Symptoms.—The symptoms are tinnitus, giddiness, deafness, unsteady or uncertain gait, and sometimes nausea and vomiting. This condition is often confounded with Ménière's disease, in which there is complete and sudden loss of hearing.

Paresis and Paralysis of the Auditory Nerve.—**Origin.**—It may be of *angioneurotic*, *rheumatic*, or *hysterical* origin.

Labyrinthitis.—There are various forms of labyrinthitis, namely: acute or chronic suppurative; acute or chronic serous. The above forms may be circumscribed or diffuse. Probably the most serious form is the acute diffuse suppurative labyrinthitis which is characterized by total deafness of the affected ear, and the various manifestations of disturbed equilibrium.

Pathology.—The labyrinthitis is nearly always secondary to an infection of the middle ear. The microörganisms present in the labyrinth are usually the same as are responsible for the middle-ear infection. In rare instances the labyrinthitis is secondary to the acute infectious disease such as mumps and cerebrospinal meningitis. The various avenues of infection from the middle ear are:

(*a*) Fistula of the horizontal semicircular canal.
(*b*) The oval window.
(*c*) The round window.
(*d*) Fistula of the promontory.
(*e*) Fistula of the cochlea near the Eustachian orifice.
(*f*) Fistula of the facial canal.
(*g*) Through the vascular or lymph stream.

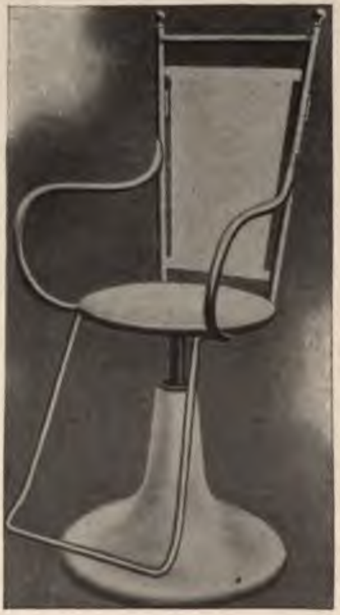

Fig. 80.—Revolving chair for testing nystagmus.

Symptoms.—In the acute forms of labyrinthitis the symptoms are impaired hearing (circumscribed labyrinthitis) or total deafness (diffuse labyrinthitis) of the affected ear, tinnitis, vertigo, nausea, vomiting, spontaneous nystagmus, loss of bone conduction and of hearing for the tones of the upper register.

In the chronic forms of labyrinthitis all symptoms may be absent other than the deafness and the chronic otorrhea.

As the cochlea is concerned only with hearing and the vestibule and semicircular canals (static labyrinth) functionate only in regard to equilibrium it can readily be seen that the symptoms of a labyrinthitis will vary according to the location and extent of the lesion.

In a diffuse suppurative labyrinthitis both functions are lost for the affected ear. In a circumscribed labyrinthitis of the cochlea the symptoms may be those of a nerve irritation (tinnitus, etc.), rather than nerve destruction (total deafness). In a circumscribed labyrinthitis of the static labyrinth the effect may be an abnormally stimulating one in which case the nystagmus is directed to the side of the diseased labyrinth (stimulation disharmony). If the static labyrinth is totally destroyed, the effect is not a stimulating one, but a destructive one (destructive disharmony). In this case the nystagmus is directed to the normal ear.

Nystagmus may be defined as a horizontal, vertical or rotatory motion of the eyeballs, consisting of two movements, a slow movement in one direction and a rapid return movement in the opposite direction. The direction of the nystagmus is indicated by the quick component.

Treatment.—Many cases recover without treatment. In certain of the diffuse suppurative cases where all function has been lost, some form of the labyrinth operation is indicated.

Syphilis of the Internal Ear.—It usually occurs as a *tertiary manifestation.* There are infiltration of the membranous labyrinth, fixation of the foot plate of the stapes, and condensation of the periosteum of the vestibule. It may be associated with a middle-ear catarrh, attended by thickening of the drum-head. There is frequently a shortened bone conduction for the middle forks in the primary or secondary stage, even though the air conduction be normal.

Symptoms.—There are sudden deafness and tinnitus, with more or less vertigo and unsteady gait. Pain is usually absent.

Prognosis.—The prognosis depends upon whether it is congenital or acquired, and recent or of long standing. The congenital type is less favorable, and the more acute cases are more favorable.

Treatment.—Potassium iodide internally, inunctions of mercury, and salvarsan are of value in recently acquired cases.

Ménière's Disease.—Symptoms.—This is an extravasation of blood into the labyrinth. It is characterized by sudden total deafness, followed by giddiness, nystagmus, great tinnitus, vertigo, staggering gait, and vomiting. There may be premonitory symptoms, as pallor, cold perspiration, etc. Bone conduction on the affected side is very much diminished or entirely absent. Upon examination, the drum-head appears normal, unless it happens to have been affected in the course of a middle-ear disease. The body inclines to the affected side in walking. The giddiness and staggering gait disappear slowly. The tinnitus is usually quite persistent and distressing. The deafness rarely improves, and if at all, but slightly.

Treatment.—This seems to be of little value. Rest and quiet, with cold applications to the head and calomel catharsis perhaps exert a slight favorable influence. Injections of 4 or 5 drops of a 2 per cent. solution of pilocarpin through a Eustachian catheter is one of the best remedies, although its use is not always unattended with danger. Those suffering with a weakened heart from any cause will tolerate its use badly. The remedy should be used once daily for a month. The patient should remain in bed and be placed upon a light diet. Quinine, potassium bi-iodide, potassium bromide, and the dilute hydrobromic acid in 15-minim doses, relieve the giddiness. If the hemorrhage be due to arteriosclerosis with high blood-pressure, the treatment should be directed to the cause.

"Ménière's Symptoms."—In the course of exudative middle-ear catarrh, chronic dry catarrh with adhesions and contractions may force the stapes into the oval window and give rise to several of the symptoms of Ménière's disease.

The two conditions are distinct pathologic entities, and should not be confounded. "Ménière's symptoms" are not attended by complete sudden deafness and ear disease is present to account for the symptoms. Inflation of the tympanum is usually followed by marked relief or the disappearance of the deafness and other symptoms of the "Ménière group."

Boiler-makers' Deafness.—This is characterized by deafness with impairment of bone conduction. A change of occupation is sometimes followed by improvement.

Senile Deafness.—There is nerve deafness with impaired bone and air conduction, and lessened ability to hear the high notes of Galton's whistle or of the König rods. The deafness may also be due to thickening or atrophy of the drum-head, and to collapse of the cartilaginous meatus.

Deaf-mutism.—**Definition.**—Absence of the power of speech on account of congenital or acquired deafness is called deaf-mutism. If it is acquired, it occurs in the early years of infancy and childhood. If the child is learning to talk, it may or may not retain the known words.

Etiology.—Congenital deafness may be the result of arrested or imperfect development of the temporal bone. Acquired deafness in infants is usually the result of the exanthematous fevers, meningitis, cerebrospinal meningitis, injuries, and syphilis.

Examination.—It is difficult to examine deaf-mutes on account of their age and lack of training. To test the hearing, the physician should stand where he cannot be seen by the child, and clap his hands loudly, ring a bell or gong, or blow a loud whistle. If the child hears, he will turn toward the noise or manifest by the expression of the face that he hears the sound. By placing the higher tuning-forks upon the vertex, the author has in some cases noted a pleased smile come upon the face; while the lower (128 v.) ones caused a displeased look. Patient and continued effort will usually enable the physician to determine the state of the acoustic nerve. In idiots it is impossible to fix the attention.

The graphophone with the ear-tubes may be used for testing mutes.

Prognosis.—Unless the power of speech is well developed when deafness occurs, the child is likely to lose his speech. If deafness occurs after the seventh year, the child generally retains the faculty of speech. More particularly, the prognosis depends upon the age of the child, whether the deafness is congenital or acquired, and upon the specific cause or causes of the condition. Acquired cases are occasionally benefited by treatment, while the congenital type is rarely improved. The more recent the deafness, the more favorable the prognosis.

If the child is intelligent, and is sympathetically, intelligently, and systematically managed, there is a possibility of teaching it many useful things, and of giving it a trained and cultured mind. The case of Helen Keller is a remarkable instance illustrative of this point. The child was not only a deaf-mute, but was blind also.

Treatment.—This should have a twofold purpose, namely: (*a*) the removal of the cause and improvement of the deafness; and (*b*) the education of the child.

All catarrhal, adhesive, and other middle-ear diseases should be most carefully treated, and the nasopharynx should also be freed from disease and the presence of growths and thickenings. Improvement in the faculties of speech and hearing does not always occur, but there may be an improvement in the nervous symptoms and the power to fix the attention.

It may be impossible to improve the hearing, while it is quite possible to improve the receptive powers of the mind. This phase of the subject is too often overlooked in the treatment of such cases. No pains should be spared to afford the much handicapped child every possible chance for mental culture and training. Schools and homes are now provided for the training and treatment of such cases, and there is a much more hopeful prognosis for them in consequence.

Deaf-mutes and the hard-of-hearing should be taught **lip reading**, as the ability to understand conversation from the movements of the lips does much to make life more enjoyable for the deaf.

EAR DISEASE AS AFFECTING LONGEVITY.

The casualties attending ear disease may be studied under two captions, namely: (*a*) accidental death on account of deafness, dizziness, and tinnitus; and (*b*) death due to the pathologic processes attending the diseases.

Accidental death may result from (*a*) *deafness*, as the one thus affected cannot hear the approach of railway trains, street cars, and other moving objects. Many deaths occur annually on this account; hence marked deafness is considered a bar to the issuance of life insurance. One affected by (*b*) *tinnitus* may confound the subjective noises in his head with those of an approaching railway train or other moving objects, and thus subject himself to considerable risk of death from traumatism. (*c*) *Dizziness*, or *aural vertigo*, may lead to accidental death by confusing the mental faculties, or by causing prostration of the patient at a time or place which places his life in jeopardy.

As most ear diseases are attended by one or more of the foregoing phenomena, their importance in estimating life expectancy becomes very great. Mild deafness and tinnitus should not, however, be regarded as sufficient grounds for the rejection of an applicant, unless the history of the case shows rapid progress of the disease. Stricture or other stenosis of the Eustachian tube, dry and adhesive catarrhs of the middle ear, chronic and acute suppurative inflammations, perforation of the drum-head, and cholesteatoma, etc., are attended by all these phenomena, and should receive most careful consideration in estimating the life expectancy. Inflammation of the auricle and external auditory meatus, eczema, and inspissated cerumen cause temporary slight deafness and tinnitus, and have little significance in this connection except as the eczema or inflammation may point to more serious involvement of the tympanic and mastoid cavities. Ankylosis and adhesions of the foot plate of the stapes cause deafness and pronounced tinnitus. Inflammation and degenerative changes of the membranous labyrinth also cause marked aural disturbances, and may therefore lead to death from accidental causes. It may be said, in a general way, that diseases of the labyrinth are attended by more pronounced disturbance of the auditory functions than disease of the middle ear, and their presence should be regarded as more grave on that account.

One year of freedom from the disease after treatment or operation should entitle the applicant to another chance to pass the examination for life insurance.

Diseases of the Ear which May Affect Longevity.—The following classification is arbitrary, and should be regarded as suggestive rather than final. The first class will include those diseases which *should not bar* the applicant from receiving the benefits of life insurance; the second class includes those diseases which render the applicant a *doubtful risk;* and the third class, those diseases which render him *an unsafe risk.*

Diseases which Should Not Bar the Applicant from Life Insurance.

1. Malformations of the auricle. 2. Benign tumors of the auricle. 3. Eczema. 4. Furuncles. 5. Cerumen. 6. Otomycosis of meatus and membrana tympani. 7. Low perforations of the drum-head. 8. Calcareous deposits in the drum-head. 9. Mild non-progressive chronic otitis media. 10. Mild deafness from tubal catarrh. 11. Anemia of the labyrinth, as a local expression of a mild general anemia. 12. Hysterical deafness. 13. Diplacusis.

Diseases which Render the Applicant a Doubtful Risk.

1. Rapidly increasing deafness from any cause. 2. Pronounced stenosis of the Eustachian tube. 3. Simple hyperemia of the labyrinth if it is due to pulmonary, renal, or cardiac lesion, is grave, and should relegate the applicant to the third class. 4. Perforation of the membrana flaccida (as it is a sign of attic, and possibly of antral and mastoid disease).

Diseases which Render the Applicant an Unsafe Risk.

1. Auricular erysipelas. 2. Gangrene and phlegmon of the meatus and auricle. 3. Perforation of the membrana flaccida attended by a foul-smelling, dirty discharge. 4. Abscess beneath the mastoid periosteum. 5. Inflammation and bulging of the postsuperior wall of the meatus. 6. Chronic per-

foration in the postsuperior quadrant of the drum-head. 7. Cholesteatoma. 8. Recurrent pains over the mastoid with chronic pus discharge from the meatus. 9. Aural polypi and granulations. 10. High perforation of the drum-head. 11. Pin-hole perforations at the margin of the drum-head (significant of tubercular disease). 12. Necrosis of the ossicles. 13. Chronic suppuration with destruction of the entire drum-head, the remnants of the ossicles being buried in a mass of granulation tissue in the floor of the attic. 14. Malignant disease. 15. Tubercular disease of the tympanum. 16. Stenosis of the external meatus following chronic discharge or the mastoid operation. 17. Ankylosis of the foot plate of the stapes. 18. Anemia of the labyrinth due to tuberculosis. 19. Ménière's disease (much more grave). 20. All inflammations of the labyrinth either from extension from the middle ear or from syphilitic, tubercular, or exanthematous infections. 21. Malignant neoplasms.

THE NOSE.

THE EXTERNAL NOSE.

ANATOMY AND PHYSIOLOGY OF THE NOSE.

THE supporting framework of the external nose is formed by the two nasal bones, the frontal processes (processus frontalis maxillæ) of the maxillary bones, the cartilaginous and the anterior edge of the cartilaginous septum. The cartilages (Fig. 81) of the nose consist, on each side, of an upper plate, known as the lateral nasal cartilage, a lower plate, known as the greater alar cartilage, and one or more sesamoid cartilages placed between the two larger cartilages. The median portion (crux mediale) of the greater alar cartilage extends along the free edge of the anterior inferior portion of the cartilaginous septum, dividing the nasal orifices. The cartilages are closely attached to each other and to the nasal bones by strong connective-tissue fibers.

The alar muscles consist of two sets, the dilaters comprising the **dilatator naris anterior** and **posterior**, the **pyramidalis nasi** and the **levator labii superioris alæque nasi**, and the constrictors comprising the **compressor nasi**, the **compressor narium minor**, and the **depressor alæ nasi**.

The Nasal Fossæ.—The Nasal Septum.—The septum divides the nose into two cavities or chambers, the right and the left. The septum (Fig. 82) is formed superiorly by the perpendicular plate of the ethmoid, anteriorly by the septal (triangular) cartilage, Jacobson's cartilage and the membranous portion of the septum separating the right and left vestibules of the nose, and inferiorly and posteriorly by the vomer, the maxillary crest, the palatine crest, and the sphenoidal crest.

The Floor.—The floor of the nose is formed by the palatal process of the superior maxillary and the palatal process of the palate bones.

The Roof.—The roof from before backward is composed of the nasal bone, the nasal process of the frontal, the body of the ethmoid, and the body of the sphenoid. The lamina cribrosa or the cribriform plate of the ethmoid which forms the major portion of the roof of the nose transmits the filaments of the olfactory nerve as it descends from the under surface of the olfactory bulb to its distribution in the mucous membrane covering the upper portion of the superior turbinal and a corresponding portion of the septum.

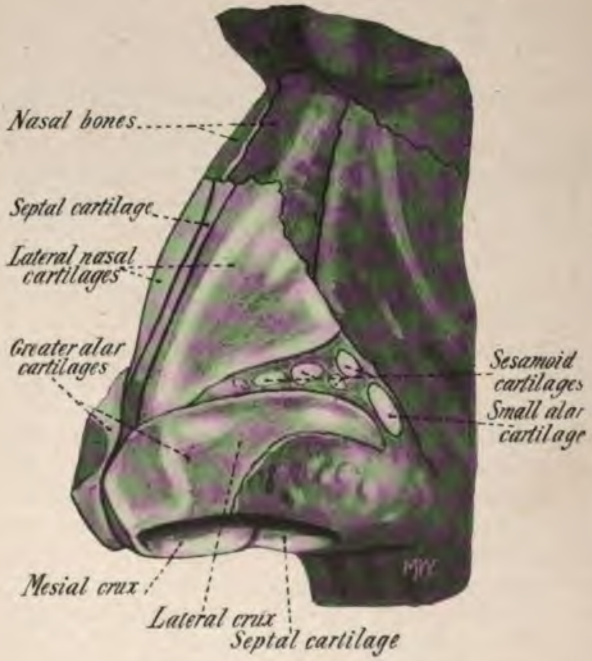

FIG. 81

The External Wall.—The outer wall of the nose is formed by the nasal process and internal surface of the superior maxilla, the palate, lacrimal, and sphenoid bones and orbital plate of the ethmoid.

The nasal fossa is divided into three meati by the three turbinates, the space situated between the inferior turbinate and the floor is called the inferior meatus, the space between the superior turbinate and the inferior turbinate is known as the middle meatus and above the middle turbinate is the superior meatus. Occasionally a fourth turbinate (supreme turbinate) is observed. The superior and supreme turbinates originate from the lateral mass of the ethmoid. The middle and inferior turbinates are attached to the superior maxilla and to the palate.

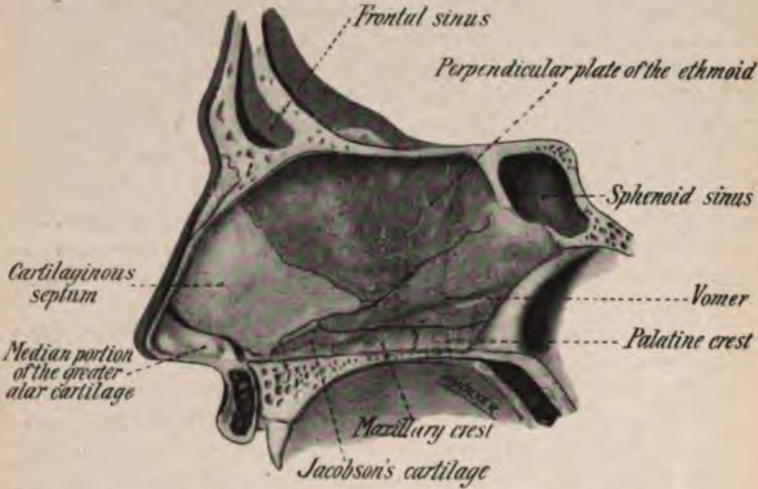

Fig. 82.—The nasal septum denuded of its membranes.

The superior meatus or ethmoidal fissure is a narrow slit-like space situated between the septum and the lateral mass of the ethmoid. The posterior group of ethmoid cells drain by one or more orifices of variable size into the central portion of the meatus. Above and behind the superior turbinate and in front of the body of the sphenoid is the spheno-ethmoidal recess, into which opens the sphenoid sinus. The middle meatus, a much more roomy space than the superior meatus, contains the orifices of the frontal and maxillary sinuses and of the anterior group of ethmoid cells. Hidden by the anterior half of the overhanging middle turbinate and situated on the external wall is a deep crescentic groove, the *infundibulum*. The crescentic opening or fissure leading from the middle meatus into the infundibulum is called the *hiatus semilunaris*. The inferior and median wall of the infundibulum forms a shelf-like ledge known as the *uncinate process*. Above the infundibulum is a hemispherical prominence, the *bulla ethmoidalis*.

The orifices of the frontal sinus, maxillary antrum and anterior ethmoid cells usually drain into the infundibulum. The frontal sinus and anterior ethmoidal cells usually drain into the anterior upper portion, and the maxillary sinus drains posteriorly to the frontal sinus. However, certain ethmoidal cells have openings located above the bulla ethmoidalis, and the frontonasal duct sometimes has an independent orifice anteriorly to the infundibulum.

The inferior meatus, the largest of the three; contains the orifice of the nasolacrimal duct located on the lateral wall from 3 to 3.5 cm. behind the posterior margin of the nostril.

The *posterior nares or choanæ* through which the nasal fossæ and the nasopharynx communicate are oval-shaped apertures located one on each side of the nasal septum. Each opening is formed inferiorly by the horizontal plate of the palate bone, internally by the vomer, superiorly by the vaginal process of the sphenoid and the ala of the vomer, and externally by the pterygoid plate of the sphenoid.

The Accessory Sinuses of the Nose.—The nasal sinuses are eight in number, four on each side of the nose: the right and left frontal sinuses, the right and left ethmoids (anterior and posterior), the right and left maxillary sinuses (antra of Highmore), and the right and left sphenoid sinuses. The sinuses are lined with the nasal mucous membrane, all are filled with air and all communicate with a nasal fossa through their various ostia.

The Frontal Sinus.—The frontal sinus varies greatly in size and form and in many instances the sinus differs in extent and shape from its fellow. One sinus appearing to develop at the expense of the other, occasionally the sinus is entirely wanting. The sinus is not present at birth, first appearing about the seventh year and attaining its full size after puberty. Bony septa may partially subdivide the sinus into one or more compartments. It communicates with the middle meatus of the nose by means of the frontonasal duct, which passes downward and backward and opens into or near the upper portion of the infundibulum.

The Ethmoid Cells.—The ethmoid cells are a series of pneumatic cells of variable size and number situated in the upper part of the nose between the orbit and the outer wall of the nasal fossa. Two groups of cells may be differentiated: an anterior group, which drain into the middle meatus and a posterior group which drain into the superior meatus. Frequently one or more ethmoid cells are found in the middle turbinate (concha bullosa). The bulla ethmoidalis is formed by ethmoid cells usually of the anterior group. Frequently a large ethmoid cell projects into the frontal sinus, producing what is known as a bulla frontalis.

The Maxillary Sinus.—The maxillary sinus or the antrum of Highmore, the largest of the nasal accessory sinuses, is an irregularly shaped pyramid with its base presenting to the nasal fossa and its apex lying in the zygomatic process of the maxilla.

The median wall or base of the antrum is formed by the vertical plate of the palate bone, the uncinate process of the ethmoid, the maxillary process of the inferior turbinate and a small portion of the lacrimal bone. The upper wall separates the cavity from the orbit. The posterior inferior wall or floor is normally the thickest and is formed by the alveolar portion of the superior maxilla and by the outer part of the hard palate. The anterior wall corresponds with the canine fossa.

The antrum communicates with the infundibulum of the middle meatus by means of a small opening, the ostium maxillare, located in the upper and anterior part of the median

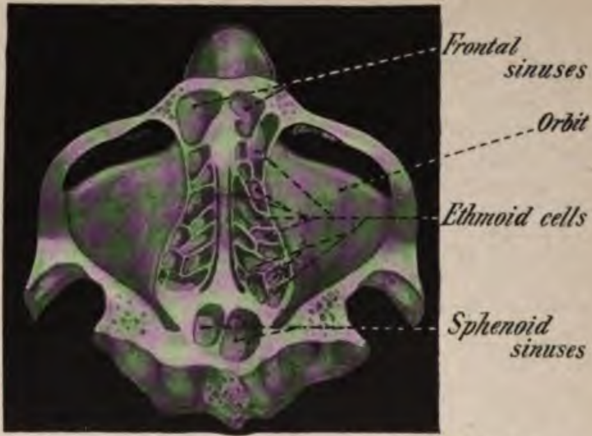

FIG. 83.—Horizontal section through the ethmoid sinuses viewed from above. (For line of section see line *a*, *a*, in Fig. 84.)

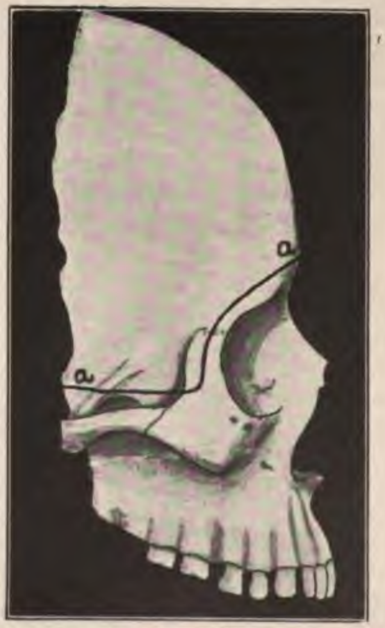

FIG. 84.—*a*, *a*, line of section through the sinuses as shown in Fig. 83.

wall or base. In a small percentage (10 per cent.) of cases an additional opening (osteum accessorium) is present, lying posteriorly to the major opening.

The Sphenoidal Sinus.—The sphenoidal sinus situated within the body of the sphenoid bone, is variable in size and often in shape. It is separated from its fellow by a thin bony partition or septum which frequently deviates to one side or the other, producing one large and one small cavity. Each sphenoidal sinus communicates with the superior meatus of the nose by means of a small aperture which empties into the spheno-ethmoidal recess.

The superior wall of the sinus lies in relation to the cranial cavity. Sluder has called attention to the close relation of the sphenoidal sinus to the third, fourth, fifth, sixth and vidian nerves. The sphenoidal sinus is not apparent until the seventh year.

The Nasal Mucous Membrane.—The nasal fossa and the sinuses are lined by mucous membrane of the columnar type, continuous with that of the nasopharynx. The membrane is divided into two regions, the olfactory and the respiratory.

The Olfactory Region.—The olfactory region is limited to the area occupied by the middle and superior turbinate and the corresponding part of the septum. The epithelium is non-ciliated columnar, consisting of two chief types of cells, the supporting and the olfactory. Numerous serous glands of a simple tubular character are present. They are known as the glands of Bowman.

The Respiratory Region.—The respiratory region embraces the remaining portion of the nasal mucous membrane. Portions of this mucosa is thick and very vascular, as over the inferior turbinate, where cavernous tissue is present. The mucous membrane is very adherent to the underlying periosteum or perichondrium.

The mucous membrane has a stratified ciliated columnar epithelium with numerous mucous-containing goblet cells. The movement of the cilia is toward the posterior nares. The glands of the mucous membrane are very numerous, tubo-alveolar in form and largely mixed mucous in type.

Erectile Tissue.—Beneath the surface of the mucous membrane covering the lower and middle turbinated bones are large plexuses of bloodvessels. Zuckerkandl describes the mucous membrane covering the turbinated bodies as consisting of connective tissue, the outer layer covered with flat epithelium, the deep layer forming the periosteum of the turbinated bones. Between them are lymph tissue, possibly lymph glands, and an occasional tubular mucous gland. Within the lymphoid tissue there are abundant venous plexuses which he calls "swell bodies." About the plexuses are unstriped muscular fibers in abundance.

The arterial supply of the nasal cavities is from the sphenopalatine and descending palatine branches of the internal maxillary artery, and from the ethmoidal branches of the ophthalmic artery. The capillaries are divided into three sets, viz.: (*a*) those going to the periosteum; (*b*) those going to the glands; and (*c*) those going to the surface of the membrane.

Those distributed to the surface form roots which empty into the veins, together with the superficial gland capillaries. The deep gland capillaries and those of the periosteum empty into the veins, forming the so-called "swell bodies"; and the blood is then conveyed by venous channels in the periosteal surface of the membrane.

The Function of the Nasal Mucous Membrane.—Its chief functions are to humidify, temper, and filter the air. The erectile tissue is controlled by the vasomotor nervous system in such a way that the inhalation of cold, dry air causes an engorgement of the venous plexuses. In a normal nose the engorgement is not sufficient to cause nasal stenosis, but just enough to favor a more rapid transudation of serum, and an additional elimination of heat sufficient to raise the temperature of the inspired air to within one or two degrees of the body heat. During the respiration of moist, warm air, the "swell bodies" contract and throw out less heat and moisture. Ordinarily from fifteen to twenty ounces of serum are thrown out in twenty-four hours. This is absorbed by the warm and expanded air and carried to the lower air tract, where it is needed for physiologic purposes. The trachea and bronchial tubes are supplied with but few glands, and are dependent upon the moisture thrown out in the nose.

Certain morbid conditions interfere with the full performance of these functions, and lead to a train of symptoms more or less common in catarrhal conditions of the larynx, trachea, and bronchial tubes.

Mucus Secretion.—The production of mucus by a normal membrane is carried on by the epithelial cells lining the mucous glands, and the superficial cells covering the membrane. The epithelial cells of the mucous glands secrete most of the mucus, but here and there on the surface of the mucous membrane there are goblet epithelial cells which also pour out mucus. The mucus is made at the expense of the protoplasm of the cell body, the process of production being somewhat as follows: the cells swell, the centres become transparent, and the protoplasm granules are reduced to small strings or particles. The mucus thus formed is discharged and the cell body left intact; or the entire cell may disintegrate and be thrown off with the mucus.

Mucus formation is a degenerative process whether it occurs under normal or pathologic conditions. Hence, in catarrhal inflammation of the nasal mucous membrane it is produced in nearly the same manner as that just described. Normally it appears as a thin viscid fluid, while that occurring in the course of catarrhal inflammation is oftentimes thick and tenacious. The difference is due to the diminished amount of serous transudation in inflammatory diseases of the membrane. The so-called normal mucous secretion is, in reality, a mixture of serum and mucus, and the amount of the two is greater than that found in diseased conditions, although the contrary seems to be true. In catarrhal inflammation the secretion is thicker and is not so readily absorbed by the inspired current of air. It must therefore be expectorated or blown upon a handkerchief. Patients often complain of an increased nasal discharge, and that there was a time when they did not need to use a handkerchief so frequently. The explanation lies in the fact that the solid elements of the mucus are increased while the serous element is much diminished; mucoid degeneration of the epithelial cells is an increased factor, while serous transudation is a diminished factor.

The Nose as a Filter.—The interior of the nose is so constructed that the inspired current of air passes over a large moist surface of mucous membrane. The dust and other foreign particles in the inspired air are deposited and discharged into the nasopharynx or through the anterior nares. The lower respiratory tract is thus protected from irritation which the dust-laden air would otherwise produce. The germs gaining entrance on the nasal mucous membrane are frequently rendered non-pathogenic by the bactericidal properties of the nasal secretion.

EXAMINATION OF THE NOSE.

Illumination of the Upper Respiratory Tract.—The same sort of light may be used as described under Examination of the Ear. The mouth may be examined without artificial illumination or the aid of reflected light.

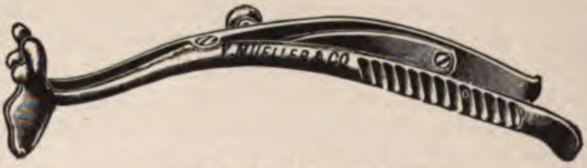

FIG. 85.—Pynchon's nasal speculum.

Instruments.—The instruments needed for this purpose are a head mirror, a nasal speculum, a probe, and postrhinoscopic mirrors ranging in size from No. 00 to No. 2.

To examine the inferior turbinal and inferior meatus, the patient should be directed to hold the head in the usual horizontal position, and not to throw it backward, as they instinctively do. The usual idea is that the interior of the nose runs upward and slightly backward. The floor of the nose runs directly backward when the head is in the normal upright position. The tip of the nose should be lifted upward with the introduced speculum so as to clear the view of the interior. With the aid of the reflected light, the **inferior turbinal** should be examined for turgescence, hypertrophy, excrescences, collapse, atrophy, crusts, and synechiæ. If the body is swollen and pits freely upon pressure, it is turgescent and not hypertrophic. If it is enlarged and does not pit upon pressure, it is either hypertrophic or the seat of some neoplasm. In hypertrophy the contour of the turbinal is nodular, while in turgescence it is rounded or globular.

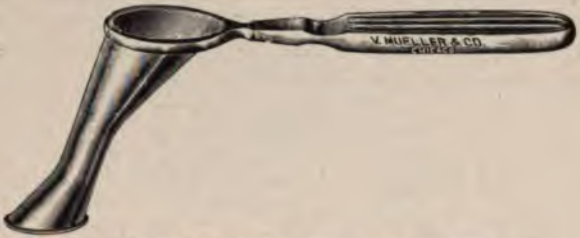

FIG. 86.—Yankauer's nasopharyngeal speculum.

The septum should be examined for spurs, ridges, exostoses, enchondroses, deflections, ulcers, perforations, mucous hypertrophies, crusts, and other morbid conditions.

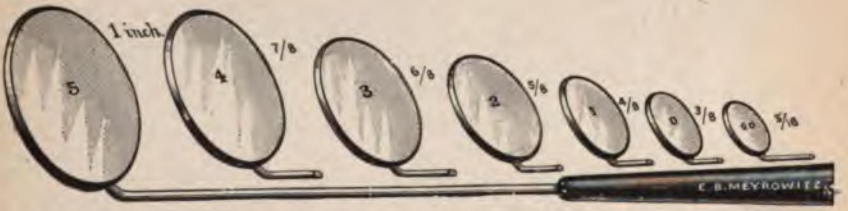

FIG. 87.—Set of laryngeal mirrors.

The middle turbinal and middle meatus may be examined by directing the patient to throw the head slightly backward and proceeding as described for the examination of the inferior turbinal. Crusts, pus, clefts, polypi, necrotic areas, and enlargement from hyperplasia should be sought for.

The superior turbinal and meatus cannot be seen except in very rare cases in which the middle turbinate is absent or atrophic. To examine such cases the head should be thrown still farther backward.

The *postrhinoscopic view* of the nose is much more difficult to obtain; indeed, in many cases it is well-nigh impossible. The palatine muscles are not habituated to voluntary control, and the thought that something is being thrust into the throat excites spasmodic contraction of the muscles.

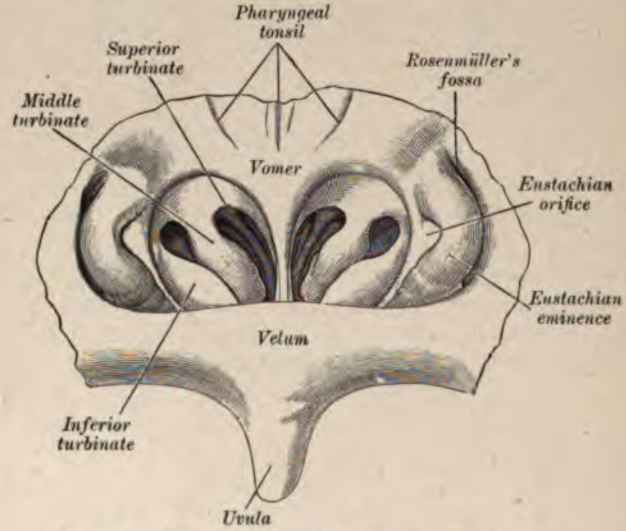

FIG. 88.—Posterior rhinoscopic image. (Schmidt.)

The tongue should be held down with a tongue-depressor and a small mirror introduced through the mouth. The mirror should be introduced on one side of the uvula, as contact with the latter excites muscular spasm. By slightly raising, lowering, and rotating the handle of the mirror, all parts of the vault of the nasopharynx and posterior choanæ are brought into view. If the soft palate is drawn upward and backward against the wall of the pharynx, the patient should be told to think of breathing through the nose.

Repeated attempts may be required to accomplish a complete examination. The condition of the vault, mouths of the Eustachian tubes, posterior ends of the middle and inferior turbinals, and the posterior end of the septum should be noted (Fig. 88).

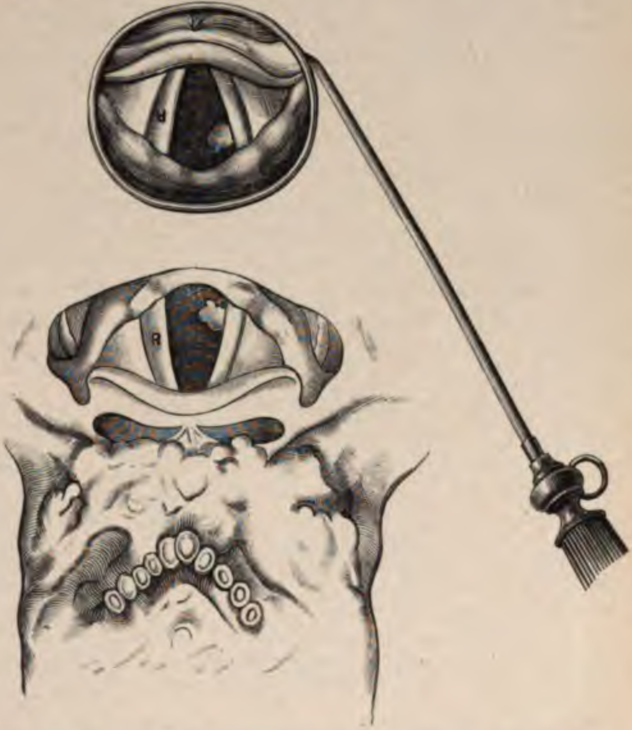

Fig. 89.—Comparison of laryngeal image with specimen. (Schroetter.)

Examination of the Base of the Tongue.—The tongue should be held as for the laryngoscopic examination and the laryngeal mirror held as for posterior rhinoscopy, except that the surface of the mirror should be directed downward. The root of the tongue is normally traversed by veins and comparatively loose folds of mucous membrane. In certain diseased conditions both tissues may be enlarged. There is lymphoid tissue here as in the vault of the pharynx, and this may be the seat of hypertrophy, although it most often occurs in adults contrary to the rule of lymphatic hypertrophy in children. The larynx may be examined at the same time by slightly lowering the handle of the mirror.

In all examinations with the laryngeal or postrhinoscopic mirrors the image is reversed.

DEFORMITIES AND MALFORMATION OF THE EXTERNAL NOSE.

Among the more common injuries, deformities and malformations of the external nose may be mentioned (1) fracture of the nasal bones; (2) twisted nose; (3) hump nose; (4) long or drooping nose; (5) saddle-back nose; (6) collapsed alæ nasi; (7) broad alæ; (8) partial loss of nose; (9) total loss of nose.

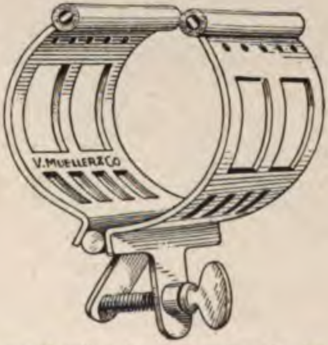

Fig. 90.—Carter's nasal splint for supporting the fractured nasal bones.

Fracture of the Nasal Bones.—If union has taken place the deviated or depressed nasal bone should be fractured from its attachment and reset in its normal position. The technic is as follows: Local or general anesthesia. An intranasal incision is made with a small scalpel through the mucous membrane of the anterior superior wall of the nose at the inferior edge of the nasal bone. A semisharp septum periosteal elevator should then be introduced through the incision and the skin and periosteum over the nasal bone stripped loose. The Steele, Asch or other stout nasal forceps should be introduced, one blade through the incision between the skin and nasal bone while the other blade remains free in the nasal cavity. The nasal bone is firmly grasped between the blades of the forceps and completely fractured from its attachments. The nasal bone should be reset in its normal position and held there by means of Carter's nasal splint (Fig. 90).

Twisted or Crooked Nose.—May be congenital or traumatic. The nasal septum is usually deviated.

The operation is best accomplished by two distinct steps, although both may be combined into one operation.

Technic.—First correct the deviation of the septum by the submucous resection (see Deviation of the Septum), leaving the cartilage forming the ridge of the nose as wide as possible.

The second step consists in fracturing and resetting the crooked nasal bones as described under Fractured Nasal Bones. This may be done two or more weeks after the submucous resection of the septum.

Hump Nose.—This condition may be corrected by an external operation or by an intranasal one.

External Operation.—An incision is made in the median line over the deformity. The skin and periosteum is elevated exposing the "hump." The projecting portion of the nasal bones or cartilage is removed by means of chisels. The skin flaps are coapted with adhesive strips or are sutured.

Intranasal Operation by the Ballenger Method.—Local or general anesthetic. Introduce a scalpel into nasal chamber and make an incision through the mucous membrane at the lower border of the nasal bone. Pass the knife between the nasal bone and the skin covering it. Separate the skin from both nasal bones with a Freer type elevator. Introduce the reverse chisel (Fig. 91) and with a downward and forward pull, parallel to the bridge of the nose, shave off the hump.

Beck's Operation.—Make incision in the anterolateral portion of the vestibule of the nose. Dissect over the hump with Mayo's scissors and with the same scissors engage and sever the cartilaginous hump. Displace the fragment in the depression (if present). A strip of adhesive plaster is drawn tightly over the bridge of the nose.

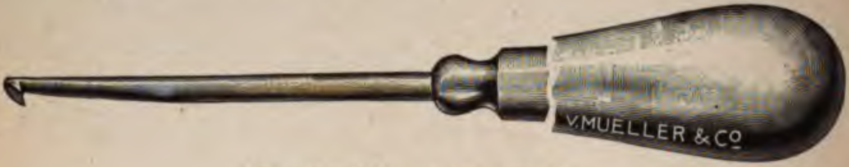

FIG. 91.—Ballenger reverse chisel.

The Long or Drooping Nose.—Kolle's Operation.—Kolle's operation consists in the removal of a portion of the under surface of the tip of the nose and bending the overhanging remaining portion down and suturing in place.

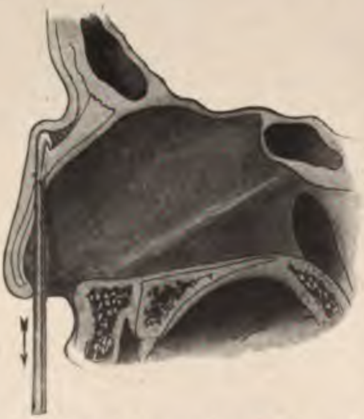

FIG. 92.—Ballenger's method of removing the "hump" from an excessively aquiline nose.

Ballenger's Operation.—Make two incisions through the mucous membrane and cartilage to the opposite mucous membrane forming a V (Fig. 93). Connect the divergent ends of the incisions at the ridge of the nose by an interesecting incision, which should separate the cartilage from the skin of the nasal ridge. Remove the triangular piece of cartilage. Retain the nose in position from four to eight days by means of a sling made of strips of adhesive plaster. The redundant skin soon contracts to the normal tension.

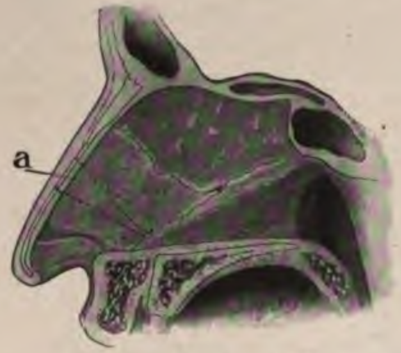

FIG. 93.—Ballenger's method of shortening a long overhanging nose. The triangular piece of cartilage (*a*) is removed *via* the nostril and the gap closed by lifting the tip of the nose upward and securing it in place with adhesive straps applied externally. At the end of four to eight days the straps are removed, union being complete.

Saddle-back or Sunken-bridge Nose.—Numerous implantation methods have been devised for the correction of saddle-back nose. Among the various substances used are gold, silver, hard rubber, cartilage, bone, fat, and paraffin. The use of the metallic or rubber devices has been largely supplanted by the use of cartilage, bone, fat, and paraffin. In the Israel operation a portion of the anterior border of the tibia is inserted into the depression through an external incision. Goodale utilizes a mucoperichondral flap, dissected from the septum and pushed into the depression beneath the skin. Carter fractures the depressed nasal bones and then suspends them in their proper position by means of his metal bridge (Fig. 90). Beck first removes the cartilaginous hump (if present) by the method given for hump nose and then uses a portion of a rib, the anterior surface of the tibia, or a portion of the septal ridge. The splinter of bone should correspond to the size and shape of the deformity to be corrected. The writer has had excellent results by using portions of the vomer inserted beneath the skin of the depression through an intranasal incision. If any depressions or uneveness exists after the transplantations, they may be smoothed over by the supplementary injection of paraffin.

Technic of Paraffin Injection.—Paraffin of a melting-point of 100° F., should be used, as that of lower melting-point seems to be more productive of accidents than that of a higher melting-point. A screw syringe is the most convenient instrument to be used. Thorough asepsis should be observed. The sterilized paraffin should be injected when solid rather than in a liquid state. The operator stands behind the patient and an assistant stands in front with his thumbs pressing firmly against the nasal bones. The operator should make sure that all air and water is excluded from the needle of the syringe. The needle is inserted above the depression and pushed beneath the skin to the most dependent portion of the depression where the thread of paraffin is slowly deposited. As the concavity is slowly filled the needle is gradually withdrawn until the upper portion of the depression is reached, when the thread of paraffin is broken by a slight twist of the syringe. The amount of paraffin injected must not be to such an extent as to produce a marked

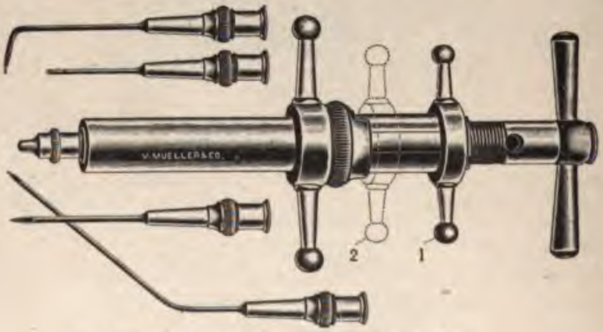

Fig. 94.—Beck-Mueller paraffin syringe.

anemia of the skin. Too small an amount of paraffin is better than too much, as subsequent injections can always be made. The paraffin is molded to the desired form during the injection.

The untoward results following paraffin injections are:

1. Pressure necrosis from too great a quantity of paraffin or from the injection of the paraffin into the skin rather than subcutaneously.

2. Infection and abscess formation when careful asepsis has not been observed.

3. Air embolism from air bubbles within the needle or syringe.

4. Paraffin embolism has been reported.

5. Diffusion or spreading of paraffin from using a paraffin of a low melting-point or when injected in a liquid state.

6. Reddened or shiny appearance of skin over area of injection has been noted and is probably due to overinjection or to injecting too near the skin.

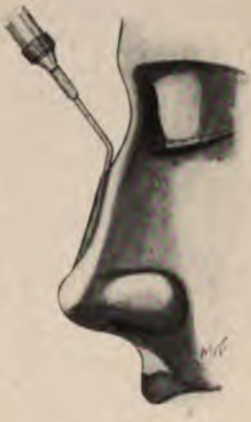

FIG. 95.—Injection of paraffin into saddle-nose.

Collapsed Alæ Nasi.—Walshaus suggests elevating a narrow strip of mucous membrane from the anterior portion of the septum, leaving the mucous membrane flap attached at its upper end, then rolling the strip of membrane into the upper angle of the nostril where it is stitched into position, forming a support for the alæ.

Lambert Lack utilizes a strip of the septal cartilage in addition to the mucous membrane. He turns up a piece of the cartilage and mucous membrane, denudes the surfaces of the cartilage and the inner surface of the ala where they come in contact and stitches the cartilage in place at right angles to the septum. A similar flap is made posteriorly to the first flap for the opposite nostril.

Paraffin is also used to stiffen the alar cartilage, but is not as satisfactory as other methods.

Broad Alæ.—Roe excises a portion of the alar cartilage with some of the subcutaneous tissue and stitches together.

Partial Loss of Nose.—Various plastic operations have been devised for the covering of unilateral defects of the nose. Legg makes a small tongue-shaped flap near the nasolabial crease, turns the flap over with the skin surface toward the interior of the nose and sutures in place. The pedicle is severed at a later date and covered with a Thiersch graft. Von Esmarch makes a similar flap but leaves the skin outward. Koenig implants a Wolfe graft from the thick skin of the back of the neck into the defect.

Total Loss of Nose.—The oldest method for correcting total loss of nose is the Italian or Tagliacozzi's method, in which a skin flap from the arm is obtained. The arm is held in place over the head until the flap is united when the pedicle is severed. Numerous modifications of this method have been devised by Israel, Nélaton, Dieffenbach and others.

By the Indian or Hindoo method a skin or a skin and bone flap is obtained from the frontal region and utilized in the formation of the new nose. Nélaton uses a strip of costal cartilage from the eighth rib and implants beneath the periosteum of a frontal flap, later (two months) turns the flap with its imbedded cartilage downward and uses the flap in the formation of the nose.

A finger is frequently used where there is total loss of the nose. The finger is first permitted to become adherent to the root of the nose and when firmly attached is severed. The denuded surface is later covered with a skin flap.

DISEASES OF THE NOSE.

Acute Rhinitis (*Acute Coryza, "Cold in the Head"*).—This is an acute inflammation of the nasal mucous membrane.

Etiology.—(*a*) It frequently results from chill or exposure. (*b*) It may be epidemic during the spring or autumn in temperate climates. (*c*) The exanthematous fevers are usually attended by an acute coryza, which is due to disturbances created within the system by the presence of the specific microörganism of the disease. (*d*) The inhalation of vapors and dust-laden air may excite it. (*e*) Chronic rhinitis and nasal stenosis are very prominent and frequent predisposing causes. (*f*) Tuberculosis and syphilis also act as predisposing dyscrasiæ. (*g*) The bacteria most frequently found are the Micrococcus catarrhalis, Friedländer's bacillus, and Bacillus influenza.

Symptoms.—The *onset* is characterized by a feeling of chilliness without rigors, lassitude, nasal obstruction, and sneezing. This is soon followed by *serous discharge* from the nose, which is quite irritating to the skin of the anterior nares and upper lip. After two or three days the discharge becomes *mucopurulent*. It may remain in this condition for a few days or weeks. The *temperature* and *pulse* show a slight febrile movement. There is *frontal headache*, due to closure of the infundibula leading from the frontal sinuses. The air thus imprisoned within the sinuses is gradually absorbed, thus producing a partial vacuum, which in turn induces engorgement of the vessels of the lining mucosa.

Treatment.—If seen in the first stage the rhinitis may be aborted by putting the patient in bed or at least indoors for a day or so, having him take a hot foot-bath, a hot lemonade and 5 to 10 grains of Dover's powder before going to bed. The local applications to the nasal mucous membrane of a 2 per cent. cocaine solution or a 1 to 5000 adrenalin solution, followed by a 25 per cent. argyrol solution will frequently stop all symptoms. A few drops of spirits of camphor on loaf-sugar, taken every two or three hours, is a remedy of some value during the *early stages* of the attack. A few drops of a 10 per cent. solution of menthol in chloroform rubbed in the palms of the hands and inhaled vigorously affords immediate relief to the nasal stenosis. A capsule containing pulverized camphor, gr. $\frac{1}{2}$; extract of belladonna, gr. $\frac{1}{8}$; and bromide of quinine, gr. j, given every hour for three or four doses; or until the physiologic dryness of the throat occurs, is also a valuable remedy in the early stage. The administration of a *saline cathartic* favors a speedy termination of the process by lowering the blood-tension, and by removing possible sources of irritation which may reside within the intestine.

In the *second stage*, or stage of mucopurulent secretion the nose should be cleared of secretion by the use of an alkaline solution used with a nasal douche. Eight grains of the chlorid of sodium to the ounce of tepid water is also a very grateful wash and diluent in this stage. The washes may be followed by inhalation of the hot vapor of the compound tincture of benzoin, ʒiv, in one-half pint of boiling water. Oily sprays containing a small amount of the oil of eucalyptus and menthol are valuable protective applications which may be used after thorough cleansing of the nares.

Should there be great engorgement of the nasal mucous membrane, the openings into the accessory sinuses may be closed. When this is the case there is danger of *acute sinusitis*. Treatment should be promptly directed to this condition, hoping thereby to abort the threatened invasion. This may be done by shrinking the mucosa of the middle turbinate with a 2 per cent. cocain solution or a 1 to 5000 adrenalin chloride solution. After the nasal engorgement is thus reduced, a 10 per cent. solution of antipyrine may be applied to prolong the period of arterial contraction.

A moderately *light diet* and slightly increased weight of underclothing will aid materially in shortening the course of the disease.

Prophylactic Treatment.—The prevention of repeated attacks of coryza should include the cure of any preëxisting nasal obstructions or infections and such measures as will restore tonicity to the vasomotor system. This latter can be best and most simply accomplished by regular exercise in the open air, and cold sponge-baths on rising, followed by friction of the skin. The skin is the largest glandular structure in the body, and has the most extensive capillary circulation, hence diminished activity of its functions is attended by auto-intoxication, which disturbs the vasomotor system In other words, if the functional activity of the skin is maintained, the tendency to take cold will be very much diminished.

Acute Dry Rhinitis.—Symptoms.—This is a modified form of the acute simple rhinitis in which there is little or no secretion, although the turgescence progresses until there is stenosis. There are frequently an intense diffused headache and pain across the bridge of the nose and back of the eyes. Objectively the middle turbinals are swollen, dry, and glazed.

Treatment.—It should be about the same as in the acute simple rhinitis. Hot fomentations across the bridge of the nose and over the eyes afford great relief to the pain.

Acute Membranous Rhinitis.—Diagnosis and Pathology.—This should not be confounded with *diphtheritic rhinitis,* in which the Klebs-Löffler bacillus is found. All membranous formations in the nose, however, should arouse suspicion of diphtheria, and most careful bacteriologic examination be made. The membrane is inflammatory in origin and is loosely attached to the mucous membrane, while in true diphtheria it is firmly attached. Cocain will neither anesthetize nor reduce turgescence.

Treatment.—The administration of iron and local alkaline washes is indicated. The false membrane should be removed daily. Insufflations of kaolin powder has been suggested.

Diphtheritic Rhinitis.—Occurrence.—It may precede, follow, or appear at the same time, as the faucial diphtheria.

Treatment.—The treatment should be as for diphtheria in the lower air tract, the nose being frequently irrigated with boric acid or other (alkaline) solution. In the chronic diphtheritic carriers, when the bacilli are obtained from the nasal mucous membrane, the frequent (every two to four hours) insufflations of kaolin for some days has rendered all cultures negative.

Acute Phlegmonous Rhinitis (*Abscess of the Septum*).—**Occurrence and Symptoms.**—This may exist as furuncles near the nasal orifice; or, more rarely, as abscess of the septum.

Abscess of the septum is usually the result of traumatism.

At first the swelling is hard, but in a few days it becomes soft and fluctuating to the touch. It is attended by local pain and redness, while the temperature curve may be slightly upward.

Treatment.—Before pus formation the local application of ice may check the process. After pus formation free incision and drainage will usually be successful. In strumous children, and in the more chronic cases, it may be necessary to curette the abscess cavity and swab it with the tincture of iodine.

Syphilitic and **tubercular rhinitis** will be considered in connection respectively with *syphilis* and *tuberculosis of the nose.*

Rhinitis of Specific Fevers.—Most of the exanthematous fevers are attended by acute rhinitis, characterized by the peculiar eruption of the disease. The nares should be frequently douched with antiseptic alkaline solutions.

Hyperesthetic Rhinitis.—*Hay fever,* or hyperesthetic rhinitis, will be described later in connection with the *neuroses.*

Acute Rhinitis of Influenza (*La Grippe*).—**Definition.**—Influenza or La Grippe may be defined as an inflammatory condition of the respiratory mucosa of the nose, throat or bronchi, which is produced by the Bacterium influenza (Pfeiffer's bacillus).

Symptoms.—The first symptoms to be noticed are a general malaise, a feeling of chilliness, with symptoms of acute rhinitis. A dryness and burning sensation of the throat is sometimes present in the early stages. The temperature frequently rises to 101° to 104° F. Aching sensations in the back and legs are frequently complained of. A dry, painful cough soon develops, with or without hoarseness.

In a few days the dry cough changes to a moist one when free expectoration from the bronchial tubes takes place.

Extension of the infective process into the middle ear and nasal accessory sinuses frequently occur.

Treatment.—The treatment should be the same as given for an acute rhinitis or bronchitis.

Vaccines are extensively used for the treatment of influenza and the results as a whole have been fairly satisfactory.

Chronic Turgescent Rhinitis.—**Synonym.**—Chronic Intumescent Rhinitis.

Pathology.—It will be recalled that along the free border of the inferior turbinated body, and its posterior and anterior ends, are located "swell bodies" which have the power to become tumefied or engorged with blood, and to collapse under certain physiologic stimuli. These are the erectile tissue of the nose. Their function is to transude serum and warm the inspired air. Under certain conditions the tumefaction may be more or less persistent, or present periods of intermission. The patient complains that at times one nostril is closed while the other is open, and *vice versa*. Or he may state that when lying upon the right side the right nostril is closed, and upon turning to the left side the order is reversed. These are the most characteristic phenomena of chronic rhinitis with tumefaction of the erectile tissue.

Etiology.—Climate seems to exert a marked influence in its promotion, as it is most common in temperate zones, and especially on the great bodies of water where the atmosphere is subjected to frequent and radical changes in temperature and humidity. It is less common in places or countries having a uniformly moist and warm atmosphere, and in those countries where the atmosphere is uniformly dry and cold. It is more prevalent in males, perhaps on account of their greater exposure to the inclemencies of the weather. Age exerts an important influence, as catarrhal inflammations occur chiefly during the period of adolescence and early adult life. It may occur, however, at any period of life. Certain occupations which necessitate the inhalation of dust-laden air or irritating vapors predispose to it. Frequent attacks of acute coryza predispose to the chronic rhinitis. Undoubtedly the most prolific source of chronic inflammations of the membrane is anterior nasal obstruction.

Symptoms.—The most prominent subjective symptom is alternating or fugitive nasal stenosis attended by a more or less profuse mucous discharge, which becomes mucopurulent when an attack of acute rhinitis supervenes upon the chronic form. The patient will "sniffle" and "snuff," or blow his nose, attempting thereby to free it from the sensation of stuffiness and obstruction. Repeated attacks of cold in the head occur, which may persist for many weeks.

Objectively, the inferior turbinate will appear as a rounded purplish-red tumor, almost filling the inferior meatus, and oftentimes touching or nearly touching the septum. If not touching the septum, strings of ropy mucus may be seen extending from it to the swollen turbinate body. Upon pressure with a probe the turgid inferior turbinate pits, thereby demonstrating its fluid contents, and proving the absence of true hypertrophy. This may also be demonstrated by swabbing the nose with a 4 per cent. solution of cocain, which after two or three minutes will cause the erectile tissue to collapse firmly over the inferior turbinal.

On account of the chronic overnutrition of the parts, there is a tendency for this form of rhinitis to merge gradually into "chronic rhinitis with true hypertrophy." Indeed, it may be truly regarded as an *early stage* of *hypertrophic rhinitis.*

Prognosis.—If the cause of the anterior nasal stenosis is removed, a cure, or at least an almost complete relief, of the distressing symptoms will usually follow.

Treatment.—The treatment is topical and surgical. The *topical* treatment consists of alkaline and antiseptic sprays, used during the morning toilet, and just before retiring at night. After thus cleansing the nose, the patient should be directed to use a spray containing 10 per cent. of the oil of eucalyptus; and 1 per cent. of menthol in liquid albolene. The treatment at the office should consist of the local application of a 10 per cent. aqueous solution of ichthyol applied on thin rolls of cotton and left in the nostril ten to fifteen minutes.

The *surgical treatment* consists of measures for the relief of the anterior nasal stenosis and for the reduction of the tumefaction of the erectile tissue. The methods of correcting anterior nasal stenosis are described elsewhere.

There are two methods of *reducing the erectile tissue,* namely: (*a*) superficial linear cauterization; (*b*) deep incisions with a knife, including the whole length of the erectile tissue of the inferior turbinate.

Superficial linear cauterization should be done as follows: Cocainize about one inch of the surface anteroposteriorly with a 4 per cent. solution of cocain applied by means of a thin film of cotton over the area to be cauterized. After a period of from five to eight minutes the cotton should be removed, when collapse and complete anesthesia will have taken place.

The cauterization is best done by the use of a straight cautery knife or electrode (Fig. 96) applied cold to the surface of greatest convexity, the electrode being moved with a sliding motion backward and forward. While the electrode is thus in motion, the electric current should be connected, and a superficial linear burn produced. The electrode should be kept in constant motion until the current is disconnected, as otherwise the eschar will adhere to the hot wire as it is removed. As the burn does not extend to the submucous tissue, but little cicatricial contraction follows its use. It seems, however, to exert a powerful influence upon the vasomotor control.

Four days later the opposite side may be likewise treated, thus alternating until the whole length of both turbinals has been cauterized. A 2 per cent. oily solution of camphor menthol sprayed into the nostril will prevent inflammatory reaction. If the cauterized line is immediately touched in its whole extent with trichloracetic acid, the reaction is limited.

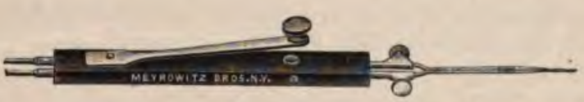

Fig. 96.—Cautery handle and electrode.

Deep Linear Incisions.—These are made along the whole length of the erectile tissue of the inferior turbinated body. Two such incisions running parallel may be made at one sitting. The parts should be first cocainized as for cauterization. Profuse hemorrhage follows, but ceases after a few seconds or minutes. If after cocainization adrenalin chlorid solution is applied, the operation will be bloodless.

The nose should be sprayed with the oily solution prescribed for home treatment, after cauterization and linear incision, and a light piece of cotton inserted into the anterior nares to prevent the entrance of dust and germs.

Chronic Rhinitis with Collapse of the Erectile Tissue.—Etiology.—It is doubtful if this condition should be classed as a true rhinitis. It is a symptom most often found in the anemias and tends to a spontaneous recovery when the anemia is cured. The condition is sometimes seen in gouty individuals, although this is rare.

Symptoms.—These are mainly due to the loss of the respiratory functions of the nose, such as dryness of the nasal and pharyngeal mucous membranes. There is also more or less laryngitis present.

Upon *rhinoscopic examination* the inferior turbinated body will be seen as a long ridge projecting from the outer wall of the nose, over which the mucous membrane is drawn quite closely. At first view it will strike the observer that true atrophy is present. The mucous membrane is dry, glazed, and anemic, with little or no incrustation. The condition is always bilateral. If crusts are present, they will be found upon the anterior third of the middle turbinal. The sense of smell is not affected. Ulceration is never present.

Treatment.—But little can be said in this connection. It should be directed to the removal of the causes producing the anemia. Local antiseptic washes and massage, as already described, are sometimes followed by engorgement of the collapsed tissues.

Chronic Hypertrophic (Hyperplastic) Rhinitis.—Definition.—This is a chronic inflammation of the nasal mucosa characterized by the presence of hypertrophy and edema of the erectile tissue. The etiology has already been given under Chronic Turgescent Rhinitis. The hypertrophic variety is but a more advanced stage of rhinitis with tumefaction. Indeed, the two types are often associated in the same nose, portions of the membrane having undergone hypertrophy while other portions remain in the state of turgescence.

Symptoms.—The symptoms are identical with those of rhinitis with tumefaction, with the exception that the stenosis is more constant and the secretions less fluid. *Objectively,* the inferior turbinated body appears as a more or less nodular mass, which pits but slightly upon pressure and does not collapse markedly upon the application of cocain or adrenalin chlorid. These points of difference stand out in sharp contrast to those presented in the turgescent variety.

Postrhinoscopic examination often reveals a bluish nodular conformation of the end of the inferior turbinals. They have been described as having a "mulberry appearance." As the tissue in this locality is chiefly vascular in structure, the hypertrophy takes the form of a varicosity. In other words, "the mulberry appearance" is due to enlargement or hypertrophy of the veins composing the erectile tissue. The mucosa in this locality being very thin, the bluish color of the venous blood is apparent.

Cough is sometimes present, and is due to intranasal pressure and to the presence of pharyngitis and bronchitis, which are the result of impaired nasal respiration. Chronic epipharyngitis is often present, more especially when the posterior ends of the inferior turbinals present the "mulberry" enlargements. Laryngitis may or may not be present.

Treatment.—Home treatment, as directed for chronic turgescent rhinitis, should be carried on. The office treatment should be directed toward the removal of the causes of anterior nasal stenosis and the reduction of the hypertrophic tissue. For the former, all spurs, ridges, exostoses, and septal deflections should receive appropriate surgical treatment.

The hypertrophied erectile tissue may be reduced or removed *en masse*. If the amount of hypertrophy is small and combined with tumefaction from engorgement of the erectile tissue and interstitial edema, deep cauterization may afford enough reduction to overcome the nasal obstruction. This is done as described under Chronic Turgescent Rhinitis, except the linear cauterization is deeper. The submucous tissue is included in the operation; hence cicatricial contraction will follow. If the hypertrophy is large and causes marked nasal obstruction, it should be removed with the cold-wire snare or file-edged scissors. The posterior "mulberry" hypertrophy should be removed with a cold-wire snare (Figs. 97 and 98), introduced through the anterior nares and insinuated over the growth. It should be tightened slowly, so as to prevent the occurrence of severe hemorrhage. A thin nasal splint made of tin foil, paraffin, or vulcanized rubber, introduced after cauterization, will prevent adhesions. It should be worn several hours a day for a week or more.

The use of the splint may be dispensed with if the patient can be inspected every forty-eight hours.

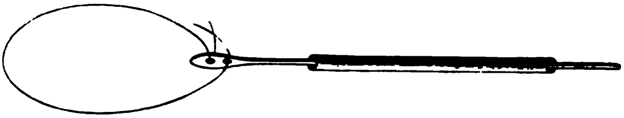

FIG. 97.—Wire threaded for a vertical loop.

Such a course of treatment is usually attended with excellent results, as not only the cause but also the results of nasal stenosis are overcome.

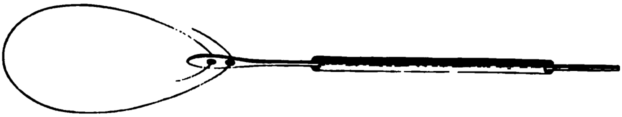

FIG. 98.—Wire threaded for a horizontal loop.

Chronic Rhinitis with Hyperplasia of the Middle Turbinate.—Pathologic Considerations.—In true hypertrophy there is an increase of all the elements composing the normal tissue, while in hyperplasia there is an increase in one of the elements, usually the connective tissue. So the term hyperplasia as here used means a connective tissue or fibrous thickening of the mucosa. There is a tendency to polypoid degeneration of the hyperplastic membrane. It is probably due to obstructed drainage from the ethmoid cells. Ulceration frequently occurs within the ethmoid cells, the bone becomes carious, and granulations and polypi form. This explanation does not, however, adequately explain all cases. It would more nearly apply to those cases of polypi which are not true myxomata, but are edematous granulation tissue. The changed character of the secretion in the ethmoid cells, together with its retention and absorption, perhaps produces an irritation and disturbance of trophic processes which lead to myxomatous new formations.

In the simple or uncomplicated form of hyperplasia there often coexists a simple catarrhal sinusitis or ethmoiditis. At a later period there is often a suppurative sinusitis associated with it.

Symptoms.—In simple hyperplasia the symptoms are not pronounced. They are chiefly referable to intranasal pressure from the encroachment of the middle turbinals upon the nasal bones and septum. This "squeezing in" process gives rise to neuralgic pains and a sense of fulness across the bridge of the nose, which may be somewhat widened and slightly sensitive to touch or pressure. There is a catarrhal secretion from the mucous membrane which is usually not profuse, but shows a tendency to become inspissated, especially upon the anterior end of the middle turbinal. There may or may not be an associated hypertrophic rhinitis. Upon inspection the middle turbinal is seen to be large and redder than normal, the anterior end being covered with a film of inspissated mucus. It lies against the septum, which is often crowded to the opposite side. One or both sides may be involved. Sometimes one side is slightly hyperplastic while the other is markedly so.

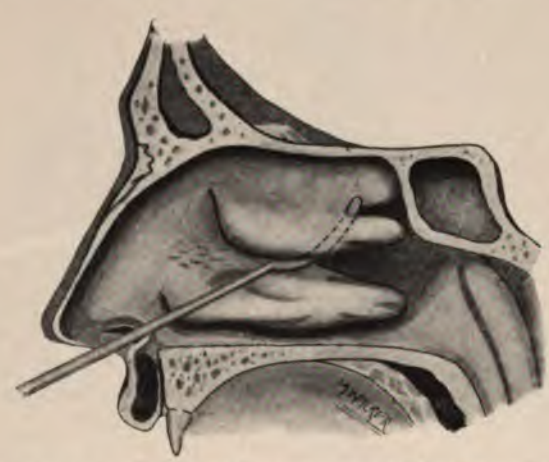

FIG. 99.—The removal of the middle turbinate with the Ballenger turbinate knife.

Treatment.—If there are no marked symptoms of pressure, surgical interference is to be deprecated. If, however, there are pressure symptoms, the enlarged portion of the middle turbinal should be removed to relieve the pressure and afford better drainage to the ethmoid cells and the mucosa of the upper portion of the nose. This may be done with a wire snare, scissors, or the Ballenger turbinate knife. The cold-wire snare may be used to remove large portions of the diseased middle turbinated body. The operation may be begun with scissors, cutting from before backward. The snare loop is then slipped through the cut, and the remaining portion removed.

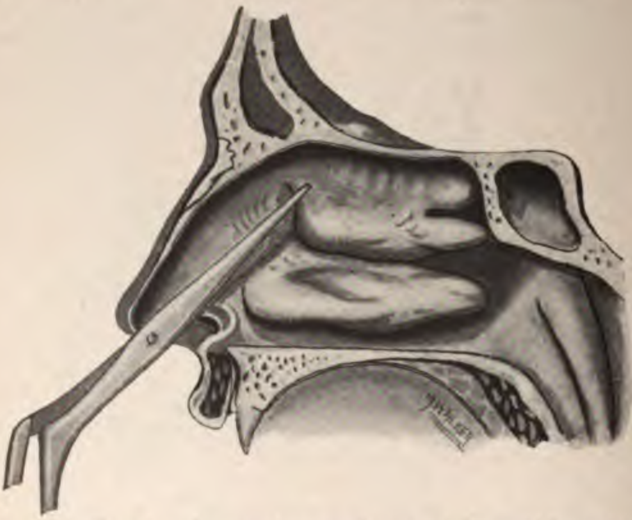

Fig. 100.—Severing the anterior attachment of the middle turbinate with scissors preparatory to the subsequent removal of the turbinate with a snare.

Chronic Purulent Rhinitis.—**Etiology.**—It is questionable whether a chronic purulent inflammation of the nasal mucosa is ever present without an accompanying purulent sinusitis, but as the presence of a sinusitis cannot always be demonstrated, the condition will be mentioned. It is believed to be due to some acute infectious disease such as the exanthemata which is acquired early in life.

Symptoms.—A mucopurulent discharge without evidence of a sinus disease is the predominating symptom.

Prognosis.—Under proper treatment the prognosis is good in the majority of cases.

Treatment.—This consists in simple cleansing and stimulating measures applied to the nasal mucosa.

Atrophic Rhinitis (*Ozena*).—**Etiology.**—There are four causes which seem to embrace most of the facts in the etiology. They are as follows:

(*a*) A *simple atrophic process* which is not dependent upon some other local disease of the mucous membrane. Meissner holds that *ozena* is due to (primitive) a broad, shallow nose and congenital development of pavement-epithelium instead of the columnar (*mucus-producing*) variety.

(*b*) *Pressure necrosis from distention* of the bloodvessels, as in chronic engorgement of the erectile tissue. This is a cyanotic congestion, the general circulation participating in the sluggish venous flow. The mucosa covering the vessels is kept upon a constant stretch; and pressure atrophy results.

(*c*) *Sclerotic atrophy* due to preëxisting inflammation in which connective-tissue cells or fibers are deposited. These after a time contract and cut off the blood supply and choke down the glandular structure of the membrane. Thus atrophy is established and functional activity diminished or destroyed.

All the causes given under the various types of catarrhal rhinitis may act as cause of the third type of atrophy. The inflammation attending them is followed by a deposit of fibrous connective-tissue cells, which cut off the blood supply and choke down the glandular tissue. The functional activity is gradually lost and the true mucous elements of the membrane finally disappear.

(*d*) Perez has discovered a bacillus which he believes is the cause of the disease.

Symptoms.—The secretions become thick and inspissated. They dry upon the surface of the membrane, where, through some change or other, they develop the *ozenic odor*. Various theories have been advanced in explanation of the odor, but none of them is proved. Among them are the following, which are suggestive if not conclusive:

(*a*) Simple decomposition of the mucopus.

(*b*) Degenerative changes in which certain fatty acids are liberated which give rise to the odor.

(*c*) The presence of certain bacteria (Perez bacillus).

Ozena as a Symptom.—Ozena is not a disease, but a sign of certain diseased conditions. Its primary meaning is a "stench," and it is in this sense that it is here used. The fetid odor is associated with an inspissated secretion, which is seen in the form of greenish crusts covering the whole of the nasal mucous membrane. There are certain other peculiar conditions associated with it, especially in those cases in which there is marked atrophy of the mucosa. For instance, the nose is frequently broad and flat, the tip somewhat elevated, and the blood anemic. The anemia is secondary and not primary. The absorption of septic material and the loss of the respiratory functions of the nose are probably the chief causes of the anemia.

The fetid odor of *syphilis* is somewhat different from that due to other causes. The odor is more offensive and the secretion richer in broken-down and necrotic *débris*. There is always bone necrosis, usually of the bony septum. It is due to the breaking down of a syphilitic gumma. The mucous membrane becomes atrophic in the later stages, and after a longer period the secretion and foul odor may spontaneously disappear and leave a comparatively clean but sclerotic membrane.

The ozenic odor of atrophic rhinitis tends to disappear spontaneously after a number of years, hence is a self-limited symptom. The mucous membrane, however, is left very much damaged. Its histologic character and physiologic function are changed or entirely lost.

Treatment.—When seen in the early stage, the treatment should aim at the removal of the causes of the inflammation that produce the sclerotic process and intranasal cleanliness.

Intranasal cleanliness is obtained by the use of antiseptic douches containing a liberal amount of mild alkalies. The alkaline antiseptic solution softens and dissolves the crusts and tenacious mucopus. A solution of 8 grains of sodium bicarbonate to the ounce of water as hot as can be borne may be prescribed for the patient's use at home.

Sometimes it is expedient to introduce cotton saturated with a 10 per cent. aqueous or glycerin solution of ichthyol. After twenty to thirty minutes the crusts will be softened and detached from the mucous membrane. They are then easily removed by blowing, and the use of forceps. This course of treatment if faithfully carried out will bring about great relief in a large number of cases.

Sugar in the form of simple syrup has been tried and found fairly successful in the treatment of the ozenal rhinitis. The theory is that cane-sugar prevents decomposition. Cotton or gauze saturated with the simple syrup is packed in the nasal cavity and left in place for one to twelve hours. A 10 per cent. solution of scarlet red in mucilage of acacia has also been recommended. Vaccines may be used.

Associated sinus disease should be treated as described under *Accessory Sinuses.*

Syphilis of the Nose.—*Primary infection* of the nose is rare. The *secondary symptoms* rarely show themselves, although mucous patches have been observed in the anterior nares.

The second stage may be manifested as an exanthema of the nasal mucous membrane, the patient seeming to suffer from an aggravated "cold in the head." In infants an obstinate catarrh should arouse suspicion of specific disease. In infantile cases there is profuse secretion which becomes dried, forming crusts in the nose. The bridge of the nose is flat.

The *tertiary stage* may come quite early, necrosis of bone having occurred at the seventh month after the primary infection. It usually occurs at from two to five years after infection.

The tertiary stage affecting the septum in adults is accompanied by little or no pain. If it affects the inferior turbinal, a boring pain is present.

Treatment.—The treatment should be about the same as for syphilis in other parts of the body. The classical remedies, mercury for the secondary stage and the iodids for the tertiary stage, should be prescribed. Gummata may be absorbed by large doses of iodid if administered before necrosis begins. In urgent cases when the time limit is short salvarsan or one of its equivalents should be used. The following prescription should be used during the administration of mercury:

℞—Potassii chloratis		gr. xc–cxx;
Glycerin		ʒij;
Aquæ	ad	℥x.—M.

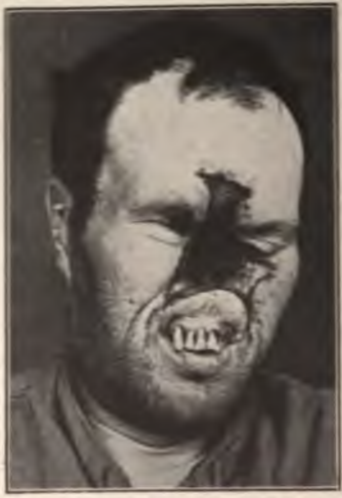

Fig. 101.—Advanced syphilitic destruction of nose. (Courtesy of Dr. G. F. Suker.)

Infants with syphilitic coryza should be fed with a bottle and the syrup of the iodid of iron, cod-liver oil, and mercury be given. The nostrils should be kept free from crusts and thick mucus by means of an alkaline spray.

Sequestra of necrosed bone should be carefully removed through the anterior nares. The horrible odor may be somewhat overcome by an alkaline douche. Where there is extensive ulceration, employ calomel fumigation by means of an apparatus consisting of a porcelain tube in which some of the powder is heated over a spirit lamp. In the meanwhile a gentle current of air should be forced through the tube with a hand bulb or compressed-air chamber. Where there is a deep ulceration the following prescription should be applied:

℞—Liq. hydrarg. nitratis . . . . ʒj–iv;
Aquæ . . . . q. s. ad ʒj.—M.

Sig.—To be applied to the sloughing ulcer of tertiary syphilis. As the application is very painful, cocain should first be applied.

Tuberculosis of the Nose.—Occurrence.—Primary tuberculosis of the nose is very rare. Involvement of the nose following tuberculosis of the lungs or throat is more common.

Diagnosis.—Microscopic examination of the tissue for tubercle bacilli should be made.

The tubercular ulcer is nodular and of a grayish color, while those of lupus are red and irritable in appearance. The ulcerative process is much slower than in syphilis. It is usually located upon the cartilaginous portion of the septum, while in syphilis the bony portion is affected.

Treatment.—This should be general and local. The general treatment should be the same as for tuberculosis elsewhere. Creosote should be given, beginning with 3-drop doses three times a day, gradually increasing the dose to 10 to 15 minims or to the point of toleration. Cod-liver oil, iron, and the syrup of the hypophosphites should be given as for general tuberculosis.

The *local treatment* consists in curettement with a sharp spoon, followed by the application of a 50 per cent. solution of lactic acid. A 5 to 10 per cent. solution of menthol in olive oil may be used locally as a palliative measure.

Tuberculin is of doubtful value.

Glanders.—Occurrence.—This disease is rare in man, but may be transmitted from the horse, in which it is common. It is a contagious disease, characterized by submucous infiltrations, or granulation tumors, which undergo rapid degeneration and discharge a foul, offensive material. The disease is essentially an acute one, but may become chronic.

Etiology.—The Löffler bacillus mallei is the specific organism which causes the disease. Horsemen or stablemen who spend much of their time with horses are especially liable to become infected. Abrasions of the mucous surface add to the probability of infection.

Symptoms.—The *acute form* or stage of the disease presents much the same clinical picture as that of most acute infectious fevers. Chills, rigors, lassitude, headache, nausea, vomiting, dyspnea, and diarrhea are usually present. Local heat, redness, swelling, and pain are present. The adjacent lymphatic glands become swollen and painful. Translucent nodules, which ulcerate, form on the surface of the mucous membrane. The pulse and temperature run high, and there are profound toxemia, exhaustion, and profuse sweating, which finally terminate in death. The close of the disease very much resembles that of typhoid fever.

The *chronic form* runs a less rapid and milder course. The symptoms are much the same as in the acute form, with their intensity much diminished.

Treatment.—The toxemia and exhaustion are somewhat modified by the administration of quinine, iron, strychnine, and whisky. Abscesses formed in the nasal or adjacent tissues should be promptly opened, their walls cauterized and cleansed frequently with antiseptic washes. Potassium iodid will modify the course of the disease in some cases.

Rhinoscleroma.—A very rare disease, characterized by the formation of firm nodular masses in the submucosa; although it may extend to the skin of the nose and adjacent structures. The disease progresses slowly by extension, is painless, and does not show a tendency to ulcerate. The nodules are composed of fibrous tissue and small round cells. There are no constitutional symptoms, although it is probably due to a microörganism.

Etiology.—It is probably of bacterial origin, the Frisch bacilli within the Mikulicz cells and in the surrounding tissue having been found. Sex, age, and local nasal conditions are not known to exert a marked influence.

Symptoms.—The infiltration nodules or plaques usually begin at the margins of the nares at the union of the skin and mucous membrane, and are firm; the skin over them is smooth, slightly shiny, hairless, and very slightly congested from venous stasis. There is no discharge, ulceration, or pain. The infiltration may extend to the skin of the external nose and face, but usually extends backward into the nares, or to the larynx in very rare instances. The nose becomes misshapen and stiff, and finally more or less stenosed. This changed appearance of the nose and the compensatory mouth-breathing give the patient quite a peculiar physiognomy. Laryngeal spasms and dyspnea may occur when the disease has advanced to the laryngeal submucosa. The uvula may become involved and subsequently atrophied.

Treatment.—Such constitutional remedies as mercury and the iodids have been tried, but with little if any effect. Total removal of the tissue is followed by its return. The only point to be gained by surgical interference is the relief of the nasal stenosis and its attendant sequelæ.

The *x*-rays and radium seem to offer the best chances for a cure.

Lupus of the Nose.—Etiology.—The etiology of this infection is closely allied to tuberculosis of the nose, but has a somewhat different clinical aspect. It is probably a chronic form of tuberculosis. Lupus of the mucous membrane of the nose is usually secondary to and associated with a similar affection of the skin of the face. The nodules are about the size of a pin-head, while those of tuberculosis are larger. The tubercular nodules tend to coalesce, while those of lupus have no such tendency. The nodules are red and irritable in appearance, while those of tuberculosis are grayish in color.

Treatment.—Tonics and potassium iodid are of some value, but energetic local treatment is more effective. Curettement, followed by the application of the electric cautery, chromic or lactic acid, is probably the most reliable method of procedure.

NASAL POLYPI.

They are usually **located** on the free border of the middle turbinal or on the outer wall of the middle meatus about the lips of the hiatus semilunaris. They rarely grow from the septum or inferior turbinated body, and if found apparently growing from the inferior turbinate, careful inspection will reveal their origin in many cases to be from the antrum. The mucous membrane of the ethmoidal cells frequently undergoes polypoid degeneration. In these cases the polypi may grow entirely from the ethmoidal cells and protrude into the nasal cavity.

The mucous membrane of the sinuses frequently undergoes polypoid degeneration.

Etiology.—Various explanations have been offered, but none of them satisfactorily explains the origin of the growths. As they are often found with suppurative ethmoiditis, some have held that all nasal polypi are due to necrosing ethmoiditis. Still others have observed that nasal polypi are preceded by an edematous condition of the middle turbinal, and have advanced the theory that they are the result of chronic edema of a hyperplastic mucosa. They are more common in men than in women, and usually occur after the twentieth year.

Symptoms.—If the growths are small, the *subjective* symptoms may be slight. If they are numerous and are large enough to obstruct the respiratory tract of the nose, a pronounced nasal tone (diminished resonance) and mouth-breathing form the chief symptoms. The patient sometimes feels the growths flapping back and forth during respiration. The nasal stenosis is sometimes complete. *Objectively* the growth appears as a pearly, grayish-blue translucent tumor, pendant from the region of the middle turbinal. It is freely movable with a probe.

Treatment.—The *surgical removal* of the growths is so simple that it seems useless to resort to other methods of treatment. The cold-wire snare affords a practical and effective method of treatment. After the application of a 5 per cent. solution of cocain to the mucous membrane of the middle turbinated body and middle meatus, the wire loop should be insinuated around the growth as near its point of attachment as possible. The loop should then be gradually tightened and the growth thus removed. Twisting off the growth with forceps is a barbarous and obsolete method of treatment which is mentioned only for the purpose of condemning it.

If the middle turbinate bone and ethmoid cells are diseased, a portion of the bone should be included in the loop and removed at the same time.

In some cases the foregoing methods are not sufficiently thorough to remove all the budding granulations and polypoid masses. The ethmoid cells should be exenterated as described under the surgical treatment of the ethmoid. The operation is not simple, and should be undertaken only after a thorough familiarity with the regional anatomy of the parts is acquired. There is some danger of penetrating the orbit, and even the cranial cavity, as the ethmoidal walls are thin and in case of chronic suppuration very much softened or necrosed.

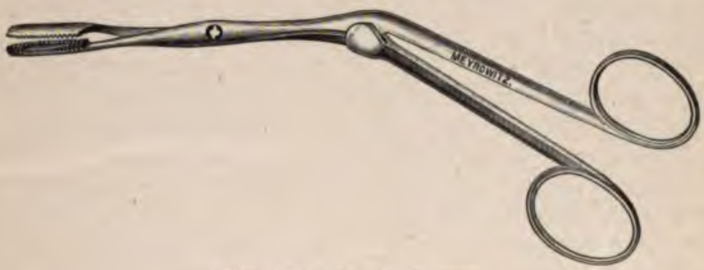

Fig. 102.—Angular forceps.

FOREIGN BODIES IN THE NOSE.

Beans, corn, peas, beads, nails, and other small articles are sometimes put into the nose by children and insane people. In one case the author removed the nipple and percussion-cap-tube, weighing 325 grains, from the nose of a man sixty years old. It had been in his nose for thirty years. The history developed the fact that thirty years ago, in firing a musket it exploded, destroying his left eye. Since that time he had been troubled with a foul-smelling pus discharge from that side of his nose. It was for this condition he was referred to me. A pair of heavy angular forceps (Fig. 102) was used in its extraction. The smaller and more usual foreign bodies may be removed with a small hook (similar to an ear hook), forceps, snare, or probe.

Rhinoliths.—Should a foreign body become encrusted with carbonate and phosphate of lime it is called a rhinolith. Mucus, a blood clot, a fragment of bone, etc., may become the nucleus of a rhinolith. The *treatment* is as for foreign bodies.

EPISTAXIS.

Etiology.—Nasal hemorrhage may be due to local causes such as traumatism, ulcers, and nasal tumors. In picking crusts from the septum or in forcibly blowing them from the nostrils the epithelial lining may be torn off and hemorrhage result. Epistaxis may also result from local congestion due to remote diseases, as chronic Bright's disease and affections of the spleen. Some of the exanthematous fevers (as smallpox, typhus, typhoid and relapsing fever), purpura, scurvy, leukocythemia, anemia, plethora, acute yellow atrophy of the liver, and phosphorus poisoning may be attended by nasal bleeding. In some cases it is due to a hemorrhagic diathesis.

Treatment.—In plethoric persons it may be well to allow the hemorrhage to continue for a time before attempting to check it. Mild astringents, as tannin, will check the hemorrhage in mild cases. Lying upon the back, raising the hand above the head, cold sponging to nose and forehead, or irrigation with water at about 110° F. are valuable remedies. The application of a 1 to 1000 adrenalin chlorid solution or a saturated solution of antipyrine by means of cotton to the nasal membrane is often followed by speedy relief. In the more obstinate cases the nose should be carefully examined for bleeding-points, which, if found, should be cauterized.

In cases which do not yield readily to the simpler methods of treatment, anterior and postnasal plugging should be done.

Anterior nasal plugging is best done with a long strip of gauze passed inward and upward between the posterior end of the middle turbinated body and the septum, where the end is held, preventing it from dropping into the pharynx. The remainder of the gauze should be packed in alternating up-and-down folds until the anterior nares is reached. This will usually check the hemorrhage, and should be left in place for from six to twenty-four hours.

Postnasal plugging is still more certain in its results. This is accomplished by passing a stout linen or silk thread *through the nose* into the pharynx by means of a Bellocq cannula (Fig. 103) or a soft-rubber catheter. The thread should be about three feet long, so that one end of it is sure to remain outside the nose. As the thread appears in the pharynx it should be seized with a pair of forceps and drawn forward through the mouth. It should then be cut loose from the cannula or catheter, which should be withdrawn. A loop should be made in the oral end of the thread and a ball of cotton the size of an English walnut fastened within it. Traction upon the nasal end of the cord will draw the ball of cotton into the pharynx, the index finger being inserted to guide it upward along the posterior pillar of the fauces into the nasopharynx. In this way the danger of inverting the soft palate into the postnares is avoided. Traction upon the cord, aided by the index finger in the nasopharyngeal space, guides the cotton into the posterior nares, where by pressure it checks the hemorrhage. If necessary, strips of gauze can also

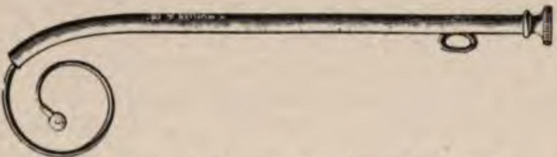

FIG. 103.—Bellocq's cannula for postnasal packing.

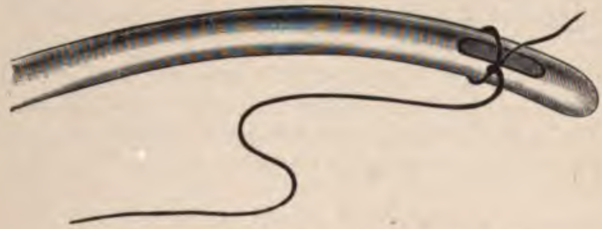

FIG. 104.—Soft-rubber catheter and thread.

be packed in the anterior nares. If the gauze is to be left *in situ* for more than twenty-four hours, it should be moistened with the compound tincture of benzoin, to prevent decomposition of the blood clot and secretions.

NASAL NEUROSES.

Anosmia, or Loss of Smell.—Etiology.—The loss of smell may be an intermittent or a permanent condition. In malaria, polypi, and rheumatic affections it may be intermittent. According to Sir Morell Mackenzie, if the anosmia lasts for two years, it is likely to be permanent. The usual causes are inflammatory conditions of the nasal mucous membrane. Thus it is often present in hypertrophic and atrophic rhinitis. Nasal polypi may cause it by preventing the current of air reaching the olfactory area. It may be due to a lesion of the olfactory centre, or of the nerve itself. It may affect one or both sides.

The **treatment** of anosmia is successful in those cases due to syphilitic inflammation, malaria, hysteria, and temporary obstructions. It is unsuccessful in old-standing cases due to inflammation of the nasal mucous membrane and in cases of central origin.

Parosmia.—Symptoms.—In this condition the patient complains of constant or recurrent perception of foul odors. It occurs in the insane, and is one of many other perversions of the senses observed among them. Persons affected by chronic nasal sinus infection characteristically complain of a foul-smelling and -tasting discharge which is odorless to others.

Treatment.—The treatment consists of appropriate remedies and surgical measures addressed to the conditions causing it.

Hyperosmia.—Definition.—This is a condition in which the sense of smell is more acute than normal.

It occurs in hysterical and very nervous persons.

Treatment.—The treatment should be addressed to the general underlying condition.

HAY FEVER (HYPERESTHETIC RHINITIS).

Synonyms.—This condition is known under various names, viz., Vasomotor Rhinitis, Vasomotor Coryza, Summer Catarrh, Autumnal Catarrh, Rose Cold, Pollen poisoning, etc.

It is characterized by annual recurrences of intense swelling of the nasal, tracheobronchial and conjunctival mucous membrane, produced in hypersensitive individuals by the sensitizing and anaphylatoxic action of the pollen of certain plants. It is attended by sneezing and profuse watery discharge from the nose. The eyes itch and burn, and are very sensitive to light.

Etiology.—The chief *exciting cause* is the pollen of certain plants. *Predisposing causes:* (*a*) a neurotic habit is perhaps the most important; (*b*) heredity; (*c*) certain morbid conditions of the nose, as hypertrophic rhinitis, deflections and thickening of the septum, polypus and hyperesthetic areas. There are, however, many cases in which there seems to be no local morbid condition. Some writers claim, however, that there are always three conditions which, acting in unison, cause hay fever; that if one of these conditions is absent, hay fever does not develop. The three conditions are: (1) The irritating pollen in the atmosphere. (2) The neurotic habit. (3) The local morbid condition of the nasal mucous membrane. If these three conditions are necessary to the existence of hay fever, the removal of either one of them will effect a cure. In many cases the conditions are all present, and in some of them the removal of the local morbid condition effects a cure. It is, however, doubtful if it should be positively stated that the three conditions are present in all cases. While much has been written upon the subject, there is yet much to learn concerning the etiology and pathology of the disease. What we now know as "hay fever" may in time be found to represent widely different conditions.

Pathology.—The special pathology of hay fever is little understood. The pollen of certain grasses, flowers, etc., and the odor of certain animals excite the train of symptoms. In some cases there is hypertrophy of the inferior turbinate bodies, especially at their posterior extremities. Polypi are often present. In others, especially those suffering from asthma, there are numerous small elevated patches on the membrane covering the septum and inferior turbinate bodies. These are found less frequently in the meati and floor of the nose. The presence of uric acid and the xanthin group of toxins in the blood is regarded by some as the chief predisposing cause of the attacks.

Symptoms.—The *premonitory symptoms* are itching, smarting, and burning sensations at the inner canthus of the eye. Some complain of similar symptoms in the throat or roof of the mouth. The onset is usually sudden, so that within a few hours the case is well established. After the premonitory stage the inferior turbinate bodies swell with great rapidity, completely occluding the nasal passages. The eyes overflow with tears, the conjunctiva becomes injected with blood, and the nose pours forth quantities of serum and mucus. Various symptoms develop from time to time, as paroxysmal sneezing, photophobia, mental depression, spasmodic asthma, etc.

The *objective* symptoms are those usually found in the various types of catarrh, septal deformities, and polypoid degeneration of the mucous membrane.

Prognosis.—This is favorable so far as life is concerned. The probability of permanent relief is good in some cases after the correction of the morbid conditions within the nose. Other cases will not improve under any form of treatment. If there is little or no local pathological change within the nares, the prognosis under treatment is less favorable than in those cases in which there is marked intranasal disease. The prognosis should always be guarded, as the neurotic element is often quite prominent and renders the chance of permanent improvement quite problematical.

Treatment.—Many remedies and methods of treatment have been advocated for the relief of this very distressing and persistent disease. None of them has proven to be of universal or even general application.

Those who favor the *neurotic origin* of the disease have advocated constitutional and diathetic remedies, with the hope that the nervous system when thus reinforced would no longer be affected by the phenomena presented in hay fever.

Others have directed their attention to the *pollen* origin of the disease, and have recommended a change of climate or the pollen vaccines as the only effective remedy.

Still others hold that *local nasal disease* is the cause of hay fever, and advocate the correction of all deformities, obstructions, and diseases of the nasal respiratory tract as the only rational method of treatment.

Clinical experience has demonstrated that in most cases all three factors must be combated in order to eradicate the affection. Oftentimes it is impossible to remove all, or even one, of the causes. In other cases the disease may be very much improved, or altogether cured, by either removing to a different climate, correcting the neurotic and lithemic habits, or freeing the nose from morbid processes.

Palliative Treatment.—During the paroxysms or acute exacerbations curative treatment should not be attempted. The distress is so great, however, that palliative measures should be administered. Sneezing, lacrimation, asthma, and nasal stenosis are the most annoying symptoms. Remedies that will overcome the nasal stenosis are chiefly indicated, as the other symptoms are secondary to it.

Cocain hydrochlorate in solution varying in strength from 1 to 10 per cent. has been used with marked relief of all the distressing phenomena. There are *three objections* to its use, however, viz.: (*a*) After a time it looses its depleting effect and the engorgement becomes greater than before. (*b*) Its effects are transient and it must be used at short intervals. (*c*) The cocain habit is often quickly formed. Hence its use should be resorted to only after trying all other methods of treatment without success.

Menthol in oily solution or suspension applied to the nasal mucous membrane and to the postnasal space is agreeable. It should be used in 2 to 10 per cent. solution. Sometimes it is essential to apply it to the posterior ends of the inferior and middle turbinals. This should be done with a curved cotton-wound applicator and the aid of the head and the postrhinoscopic mirrors.

Suprarenal extract gives temporary relief, as it rapidly and completely depletes the swollen turbinals. A 1 to 5000 solution should be sprayed into the nose every three or four hours.

Linear scarification, as described under Chronic Rhinitis with turgescence, sometimes affords relief. Cocain anesthesia should first be produced.

Severe *asthmatic attacks* are often benefited by the administration of the following mixture (Kyle) every three or four hours:

℞—Morphinæ sulphatis	gr. $\frac{1}{8}$
Strychninæ sulphatis	gr. $\frac{1}{60}$
Hyoscinæ hydrobromatis	gr. $\frac{1}{200}$—M.

If the foregoing remedies fail to afford relief, the patient should be *sent to a suitable climate* for a period of two months, or until the "hay-fever" season is over. This does not effect a cure, but affords relief from the distressing symptoms.

Interparoxysmal Treatment.—During the intervals of freedom from acute manifestations the nose and general system should be brought to as near the normal as possible. Such treatment should not be postponed until the paroxysmal seizures appear. The nasal mucous membrane is then in no condition for surgical interference, and the general system will usually not respond promptly enough to abort the attack. As there are ten or eleven months of freedom from acute symptoms, this time should be used in preparing the patient to withstand the peculiar irritation that causes the paroxysms.

Intranasal irregularities and *hypersensitiveness* should be corrected during the quiescent period. During the winter and spring months the mucous membrane of the nose is in the most favorable condition for surgical interference. Spurs, ridges, hypertrophies, hyperplasias, deflections, excrescences, polypi, etc., should be removed or corrected by appropriate surgical and remedial measures. These have been described elsewhere and will not be further considered here.

The *hypersensitive areas* should be located with a blunt probe and cauterized with trichloracetic acid or a white-hot platinum knife. The areas may be located by passing the probe over the mucosa; the moment a sensitive area is touched the patient will experience intense burning, smarting, itching sensations in the nose and inner canthus of the eye. A flat cautery point, previously adjusted so that it will come to a white heat the moment the current is connected, should be applied. The flash of white heat will make a superficial burn and will be unattended by pain. If the electrode is heated to only a cherry-red, the tissues will be deeply burned and there will be intense pain. From two to four spots may be cauterized at a sitting. After a period of four to seven days the treatment may be repeated with safety. It should be continued until all sensitive spots are removed. The posterior ends of the turbinals and septum should be carefully searched for sensitive areas, and if found should be cauterized with the aid of the postrhinoscopic mirror.

Cocain should not be used in the reduction of the sensitive areas, as it would interfere with the symptoms which enable the operator to locate them.

Pollen Extracts.—Recently the specific treatment of hay fever by active immunization with pollen extracts has been advocated. A fresh, sterile, solution of uniform potency should be used. The immunization should be begun before the advent of the hay-fever season. Manning reports 21 cases tested: 14 were objectively and subjectively relieved. Oppenheimer and Gottlieb report 11 cases, 5 were cured for the season, 4 were improved, 2 not improved. Koessler reports 41 cases (36 of these had autumnal catarrh) 4 were apparently cured, 29 improved and 8 not improved.

THE SEPTUM.

OBSTRUCTIVE MALFORMATIONS OF THE NASAL SEPTUM.

The deformities of the nasal septum may be divided into (1) spurs, ridges, or crests; (2) deviations or deflections.

Etiology.—Trauma is undoubtedly a factor in a small number of cases. Probably the majority of cases are produced by an unequal development of the septum as compared with the nasal cavity. A high arched palate is associated with many cases of deflections.

Pathology.—The malformation may involve the cartilaginous or bony portions only or both may be involved. The spurs and ridges are usually located anteriorly on the lower third of the septum at the union of the cartilaginous and bony tissues. The deviation may be "c" shaped, where the convexity produces a partial or complete stenosis on one side or "s" shaped, in which both sides may be more or less obstructed.

Symptoms.—The symptoms when present are those incident to nasal obstruction to respiration or drainage.

(*a*) Impaired breathing on one or both sides.

(*b*) Headache is frequently present if there is a contact or pressure-point between the septum and a turbinate.

(*c*) If the deviation obstructs the outflow of the sinuses draining into the infundibulum and there is a coexisting sinus disease, the various symptoms of the sinus trouble may be aggravated.

(*d*) Crusting and bleeding from the nose is frequently present and may be due to the irritation of the mucous membrane consequent to the exposed position of the deviation or spur in the air channel.

(*e*) Asthma, hay fever and neuralgia sometimes have as their reflex causes an intranasal pressure or irritation.

(*f*) An external nasal deformity is frequently present with a marked deviation of the septum.

Exostoses and Enchondroses.—**Synonyms.**—Spurs; Ridges; and Crests.

Occurrence.—These thickenings of the septum are composed of cartilaginous or bony tissue, or of both combined; and are usually located anteriorly on the lower third of the septum at the union of the cartilaginous and bony tissues. If the growth is in the form of a ridge or crest, it usually extends from the anterior nares (where it is cartilaginous) backward and upward to the bony portion of the septum (where it assumes the type of tissue from which it grows). On the bony portion of the septum isolated spurs of considerable size are often found, the presence of which cannot always be determined on account of a coexisting deflection of the septum or excessive hypertrophy of the turbinals. A spur growing in this location is often the source of considerable irritation and obstruction, and should be removed.

Treatment.—In view of the disturbed functions of the respiratory tract, and secondarily of those of nutrition and tissue metabolism, it becomes apparent that the removal of the spurs or other forms of thickening should be undertaken. Fortunately, the procedure in most instances is a simple one. The mucous membrane covering the spur or ridge should be anesthetized by the application of a 4 per cent. solution of the hydrochlorate of cocain. A thin film of cotton, large enough to cover the spur or ridge, should be moistened with the solution of cocain, and applied over the growth with a probe. It should be removed in from five to eight minutes. If the parts are not thoroughly anesthetized, a second application should be made in a similar manner.

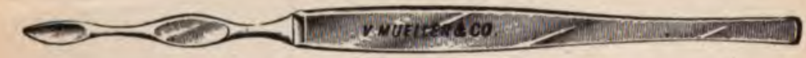

Fig. 105.—Ballenger's septum knife.

The mucous membrane covering the spur or ridge should be elevated and the flap pushed aside.

A Bosworth or Mial's saw should be introduced beneath the growth at its base, sawing from below upward, so as to leave the upper portion of the thickening unobscured by blood (Fig. 106).

At the close of the operation the periosteum and mucosa should be replaced and held in position for twenty-four hours with a gauze dressing.

When the spur is located far back on the bony septum, it is often quite dense, and may require the use of a V-shaped chisel for its removal. If a saw is used care should be exercised in the to-and-fro motion that other parts are not injured by the distal end.

Simple cartilaginous spurs of small dimensions may be removed with a knife or spokeshave. The wound remaining after these operations generally heals within a few days.

If the inferior turbinals are turgescent, a soft-rubber splint, devised by Richard Lake, or a thin sheet of celluloid or gutta-percha, should be introduced and worn until the healing process is complete, otherwise adhesions may form between the inferior turbinal and the freshly wounded surface.

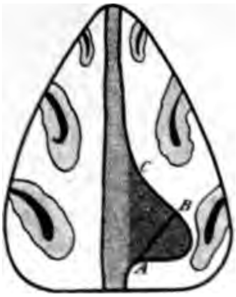

Fig. 106.—Diagrammatic representation of a spur on the left side of the septum: *A*, point at which sawing should begin; *C*, point at which saw should emerge; *A*, *B*, line along which the saw will travel *unless care be taken*. (Coakley.)

Deflection of the Nasal Septum.—**Etiology.**—The etiology of this very common condition is still open to discussion. It is safe to conclude, however, that it is sometimes due to traumatism received during childhood or at a later period of life. It is rarely congenital, and is not often seen before the seventh or eighth year. As the septum is formed by the union of the vomer below and the perpendicular plate of the ethmoid above, deflection may result from excessive development of one or both of these bones.

Symptoms. These are incident to nasal obstruction, and have been described elsewhere.

Upon *rhinoscopic examination*, the deflection may be seen as an angular or curved convexity upon one side and a corresponding angular or curved depression upon the other. It may be limited to the cartilaginous, although it usually involves the bony septum as well. It may be horizontal or vertical, or a combination of the two. Upon the side of concavity there may be compensatory hypertrophy of the turbinal bodies. When this is extensive, a portion of the compensatory tissue should be removed before undertaking the correction of the deflection, otherwise it may be impossible to bring the septum to the midline.

Treatment.—The relief which follows correction of deflections of the septum is marked, and fully justifies the surgical procedure necessary for its accomplishment. Experience has demonstrated that only a few methods of operating are followed by permanent relief, the septum often returning to its former position. In view of this fact, it is important that a proper method of operating be chosen. This will be controlled largely by the degree, type, and location of the deflection. In the vast majority of cases the submucous resection of the septum as first devised by Killian is the ideal method.

Operative measures should not be resorted to simply because the septum is deflected or thickened, but to overcome stenosis and to equalize the respiratory space within the two nostrils.

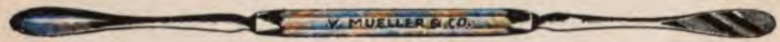

FIG. 107.—The Hajek-Ballenger submucous elevator.

The *Gleason operation* which is used very rarely at the present time consists of a U-shaped incision. It is made so as to include the whole of the deflected or bulging portion of the septum except at the top, where the legs of the U-shaped incision end. The tongue, or flap, thus formed is pushed through the opening to the concave side, where it is held by impinging upon the sharp edge of the bevelled opening. The bulging or convex surface is thus carried toward the concave side and away from the obstructed side.

The Submucous Resection of the Septum.—*Anesthesia.*—Cocain anesthesia is much more preferable, although a general anesthetic may be given. It is the writer's custom to dip a small pledget of cotton, mounted on a slender nasal applicator, into a fresh solution of adrenalin chlorid (1 to 1000), the excess adrenalin squeezed from the cotton and the moist cotton then dipped into the powdered cocain. Both sides of the septum are carefully rubbed with the mixture. Three applications, at intervals of five minutes, will usually suffice to induce complete anesthesia. If the columnar cartilage is to be removed, the writer injects a 4 per cent. novocain solution beneath the skin at the junction of the skin and mucous membrane, as the external application of cocain on the skin will not produce anesthesia.

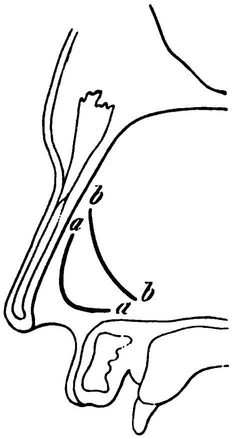

FIG. 108.—Incisions of the submucous resection of the septum; *a*, *a*, the Hajek incision; *b*, *b*, the Killian incision.

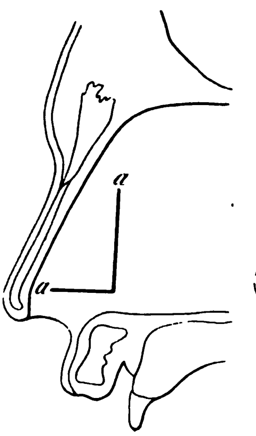

FIG. 109.—*a*, *a*, the Freer incision.

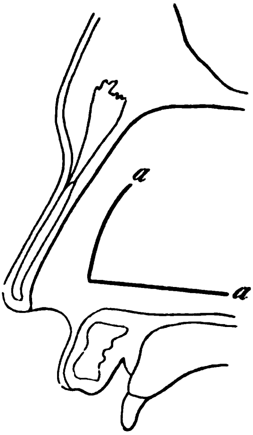

FIG. 110.—*a*, *a*, an incision which is sometimes advisable when a broad, flat wing of bone extends from the nasal crest.

Position of the Patient.—Most operators prefer the sitting position in a chair which can be easily converted into a reclining position. However, a reclining position may be used throughout the operation.

The Incision.—Many operators prefer to make the incision on the side of the convexity, believing that greater freedom of access is thereby obtained to the septum. The writer in all but most exceptional deviations involving the columnar cartilage makes his incision on the patient's left side. The

Killian incision (Fig. 108) is probably most popular although the Hajek incision (Fig. 108) may be made when the deviation involves the columnar cartilage. Freer uses a reversed L (Fig. 109) incision. The incision should extend through the mucous membrane and perichondrium to the cartilage but not through it.

The Elevation of the Mucoperichondrium and Periosteum.—To start the elevation a sharp or semisharp elevator may be used, care being exercised to get beneath the perichondrium. The cartilage may be recognized by the glistening white

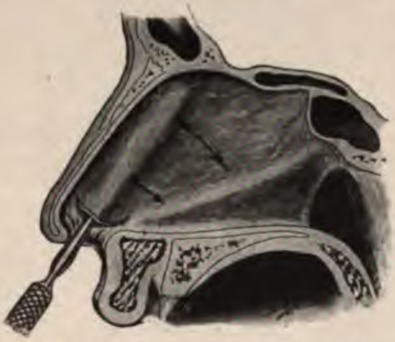

FIG. 111.—The Hajek elevator introduced beneath the mucoperichondrium along the line of least resistance. When thus introduced, the elevation should be made in the whole shank of the instrument in a downward and backward direction to the crest of the vomer. The periosteum along the crest of the vomer should then be incised.

surface as in contradistinction to the perichondrium, which has a velvety red color when separated from the mucous membrane. Having started the elevation, a semisharp or dull elevator should be introduced and the membrane elevated parallel with the ridge of the nose (Fig. 111). The elevator is now pushed downward and backward, stripping the membrane down to the crest of the vomer. The membrane should be elevated at least one-half inch beyond the deviated portion of the septum that is to be removed.

As the periosteum of the vomer is not continuous with the perichondrium, but is reflected from one side of the vomer over its crest to the opposite side, it is frequently necessary to first split the periosteum along the crest of the vomer before elevating. This may be done with a small sharp scalpel and the elevation continued with the blunt elevator.

The Incision through the Cartilage.—This may be done with a small sharp knife or by means of a small sharp curette. Extreme care should be taken not to wound the opposite membrane, as perforation is likely to result if both membranes are cut at the same point.

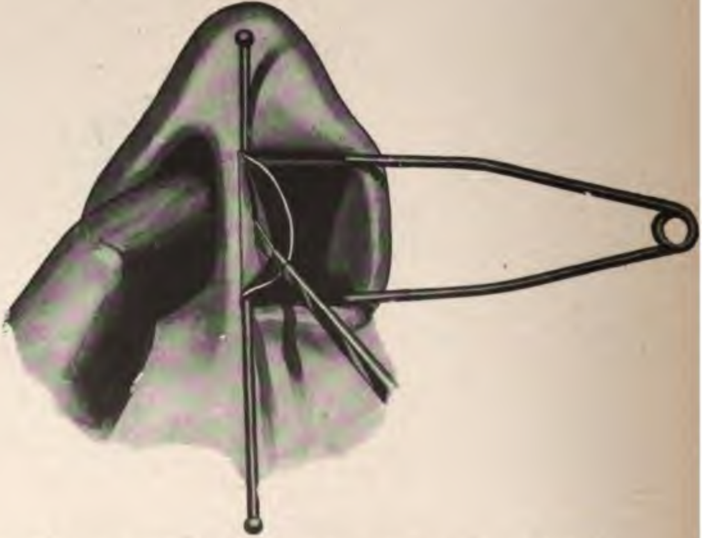

Fig. 112.—The mucoperichondrium being elevated, the cartilage is incised, care being exercised to avoid perforating the mucoperichondrium upon the opposite side of the septum.

The Elevation of the Opposite Mucoperichondrium and Periosteum.—The opposite membrane should be elevated as described for the first elevation except a semisharp or dull elevator may be substituted for the sharp instrument in starting the elevation. All elevating should be done with the shank of the elevator rather than with the tip, as the tip is much more likely to perforate or tear the membrane than the broad edge of the shank.

The Removal of the Cartilage.—This may be done with angular knives, Killian's double-edged spokeshave, punch forceps or more easily, in the writer's opinion, with the Ballenger swivel knife (Figs. 114 and 115). Before using the swivel knife the mucoperichondrial flaps may be distended from the cartilage by means of a septum speculum. The swivel knife should be applied to the cartilage at the upper portion of the incision. It is first pushed upward and backward, then downward and finally forward and out, thus encircling the portion of the cartilage to be removed. Sufficient cartilage should be left along the ridge of the nose to form an adequate support.

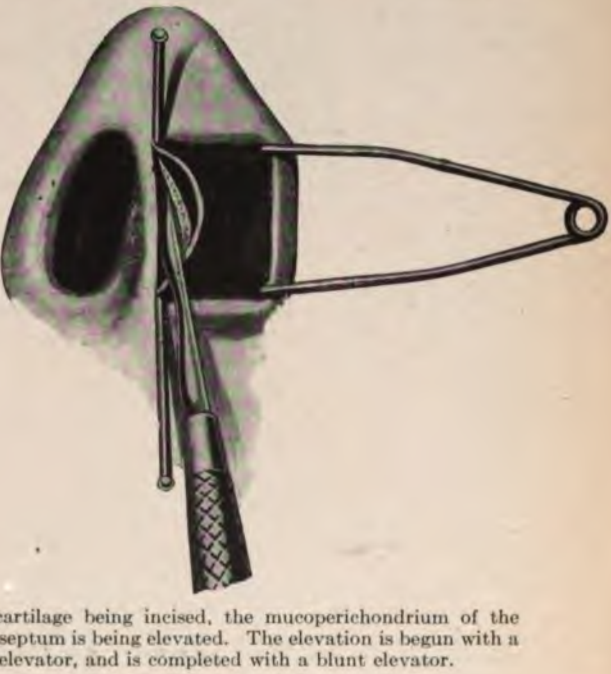

Fig. 113.—The cartilage being incised, the mucoperichondrium of the opposite side of the septum is being elevated. The elevation is begun with a sharp or semisharp elevator, and is completed with a blunt elevator.

The Removal of the Bony Septum.—The perpendicular plate of the ethmoid may be removed with the Foster-Ballenger bone forceps or any other sharp cutting forceps that may be desired. The vomer may also be removed with the bone-cutting forceps, although if the crest is greatly thickened it is necessary to use a V-shaped chisel (Fig. 117) or some heavy type of bone-cutting forceps such as that devised by Hurd. Brown uses a guarded drill driven by a motor.

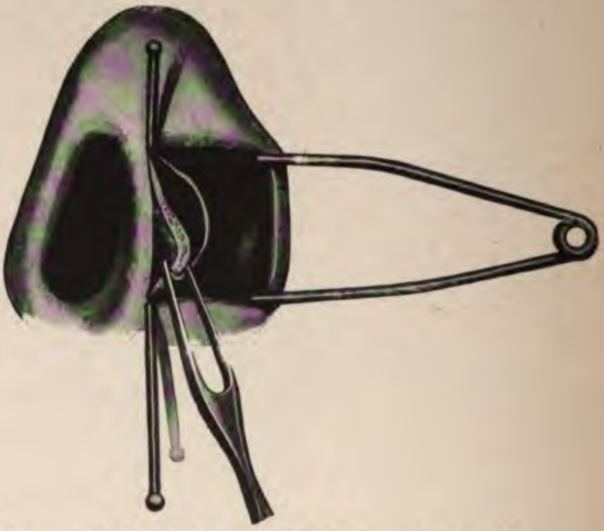

FIG. 114.—The Ballenger swivel knife in position at the lower portion of the incision of the cartilage.

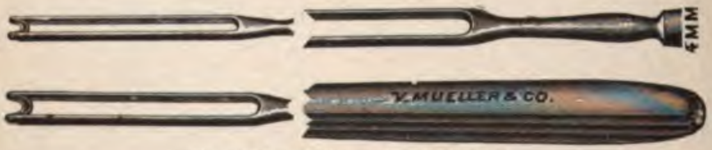

FIG. 115.—Ballenger's swivel knife.

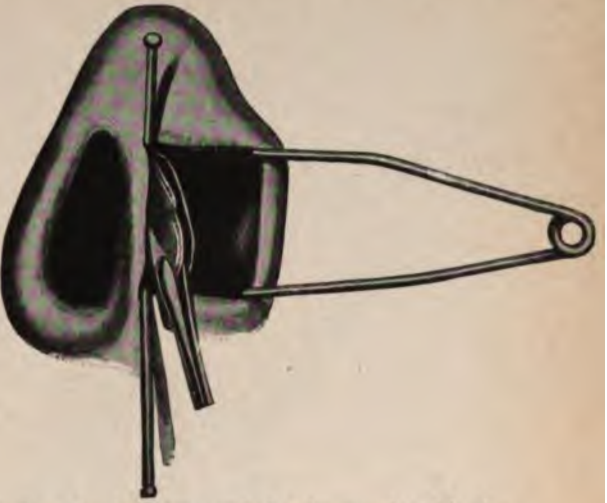

FIG. 116.—The cartilage, having been excised submucously with the swivel knife, is removed from the mucoperichondrial pouch with dressing forceps.

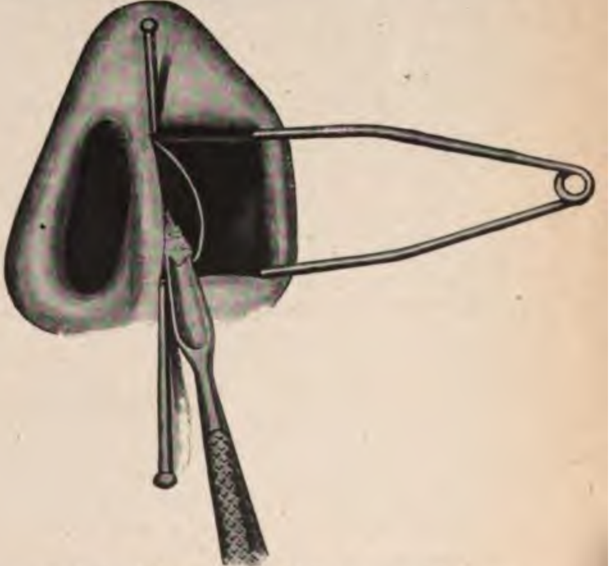

FIG. 117.—The removal of the thickened crest of the vomer with the V-shaped gouge.

The Dressing.—All of the deviated portion of the septum having been removed the membranous flaps should be coapted and a loose packing of sterile gauze or sterile gauze impregnated with vaseline placed so as to hold the flaps in their proper position and also to prevent the formation of a blood clot in the mucoperichondral pouch. A dressing, frequently used and one which has given the writer very satisfactory results is the Simpson-Bernay splint, composed of layers of compressed cotton with an external layer of linen. A few drops of sterile water or hydrogen peroxide instilled into the ends of the splints cause them to swell and compress the membranes together. The splints can be more easily removed and will not adhere to the septum if vaseline is spread over the sides that will be in contact with the septum or turbinates.

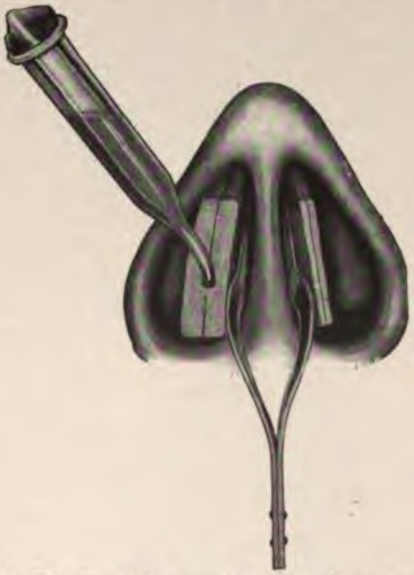

FIG. 118.—The Simpson sponge-tent dressing in position at the close of the submucous operation. The left side shows the tents dry, the right, moist and swollen. The Foster speculum holds the membranes in apposition while the tents are being introduced.

The After-treatment.—The dressing should be removed at the end of twenty-four hours and the nose cleansed of mucus and blood clots. If bleeding is excessive, the nose may be loosely packed with a fresh dressing at the point of bleeding; however, this is seldom necessary. The patient should be seen daily or every second day until all swelling and inflammatory reaction has disappeared. Frequently no after-treatment at all is required. However, the patient should be kept under observation for a few days.

Accidents.—1. *Perforations.*—This is usually due to tearing or puncturing both membranes at the same point. Probably the most common location for this mishap to occur is when the cartilage is incised. Before the operator is aware of it the incision has extended through the mucous membrane of the opposite side. When this happens, if the operator will make a new incision through the cartilage a quarter of an inch posterior to the first, no ill results need be anticipated. As the skill and technic of the operator is developed this accident can be almost entirely eliminated.

2. *Sinking in of the Ridge of the Nose.*—This accident has been reported and is probably due to one of the following conditions: (*a*) the removal of the cartilage too near the ridge of the nose; a strip of cartilage one-fourth of an inch in depth should be left for support; (*b*) chondritis following the operation due to lack of proper asepsis; (*c*) syphilis before, at time of, or after the operation; (*d*) trauma at time of or after the operation.

3. *Hematoma of the Septum.*—This is probably the most frequent complication following the septum operation and need cause the least anxiety, as a few weeks or months will usually suffice to cause a complete absorbtion of the clot. It is usually caused by improper packing.

4. *Secondary Infection of the Middle Ear or Sinuses.*—The infection may be acquired following the operation if much swelling and trauma results which temporarily interfere with the proper drainage and ventilation of the nasal cavity. The infection may be introduced at the time of operation by a lack of proper asepsis.

Nasal Synechiæ (*Adhesions*).—**Etiology.**—They may be congenital or acquired, and are composed of fibrous, cartilaginous, or bony tissue. They are usually caused by the contact and plastic union of two opposed inflamed surfaces. They may follow operations and cauterizations of the nasal mucous membrane, or they may be present in deflections of the septum.

Treatment.—Treatment by removal is required, as the band affords considerable obstruction to respiration. The band may be composed of bone tissue and require the use of the saw for its removal. In other cases it may be mucous or fibrous tissue, and may be removed with a synechia forceps.

In some cases the tendency to reunion is so strong that it is necessary to introduce paraffin or soft-rubber splints between the surfaces.

They should be worn until all tendency to reunion is over.

ULCERATION OF THE NASAL SEPTUM.

Etiology.—Ulceration is due to local irritations from causes within the blood or from external influences. A lithemic diathesis affords a local irritation, which, when the local predisposing conditions are present, lead to ulceration and perforation of the septum. It sometimes occurs in tuberculosis, syphilis, diphtheria, etc.

The *external influences* which cause it are the inhalation of dust-laden air, picking at the nose with the finger-nail, and the formation of desiccated crusts upon the anterior portion of the septum. A prominence upon the septum, as an exostosis or spur, predisposes to the formation of the crusts, the current of the air drying the mucus more rapidly here on account of its exposed location. Foreign bodies and pressure from intranasal growths may cause it. Syphilitic ulceration and perforation have long been recognized as occurring in the nose. Ulceration or perforation of the septum in the very young should excite suspicion of congenital syphilis.

Treatment.—This should be stimulating in sluggish ulcers, characterized by grayish indurated bottoms, while the more inflamed and boggy red ulcers should receive sedative applications. Mildly stimulating applications of a 4 to 8 per cent. solution of the nitrate of silver, or a 20 per cent. solution of chromic acid are suitable lotions for the grayish indolent ulcers. If these and other stimulating applications fail, the base of the ulcer should be curetted. Inflamed boggy ulcers should be treated with an ointment containing 2 per cent. of carbolic acid; 1 per cent. of menthol; and 3 per cent. of boric acid.

PERFORATION OF THE NASAL SEPTUM.

Etiology.—Congenital perforation is very rare. The causes have already been given under Ulcerations, of which perforation is but a later stage. Ulceration and perforation are due to (*a*) arrested or faulty development; (*b*) to local inflammatory action; (*c*) to traumatism; and (*d*) to some systemic infection, as syphilis, or the infectious fevers. It is most common during middle life.

Symptoms.—When large, crusts and ulcerated edges may form on the margins of the perforation and occasion considerable annoyance to the patient. Slight hemorrhages occur upon forcibly blowing the nose, or in attempting to remove the crusts with the finger-nail. If the perforation is small, a whistling noise is sometimes noticed by the patient. If the crust is large, the nose will feel stuffy.

Treatment.—These conditions can be rendered more tolerable by suitable treatment. To remove the crusts, have the patient use an alkaline solution with a nasal douche until they are softened and can be removed without disturbing the epithelium beneath. In this way hemorrhage will be avoided. Instillations of a 25 per cent. solution of peroxide of hydrogen will also soften the crusts. A 3 per cent. solution of the nitrate of silver is very useful in the cure of the ulcers or abrasions on the margins of the perforation.

Goldstein has devised a plastic flap operation in which he first freshens the rim or edge of the perforation, then elevates the mucoperichondrium for a distance of one-half inch around the edge of the perforation. He then resects a ring of cartilage from the edge of the perforation with the Ballenger single-tined swivel knife. A mucous membrane flap, larger than the perforation, is then dissected from the septum and turned into the perforation. The edges of the flap are tucked between the elevated membranes around the perforations. The flap is held in position with three or four stitches.

Jackson transplants tissue from the inferior turbinate to close the septal perforations.

The writer has had good success in small perforations from transplanting portions of the cartilaginous septum. The edge of the perforation is freshened and a rim of the mucous membrane separated around the perforation. The fresh cartilage is inserted and held in place with a loose packing. The cartilage forms a framework upon which the new-formed mucous membrane can grow.

INFLAMMATORY DISEASES OF THE NASAL ACCESSORY SINUSES.

General Remarks.—For clinical purposes the nasal accessory sinuses may be divided into two groups or series.

Group I is composed of the frontal, the anterior ethmoidal and the maxillary sinuses. These sinuses drain into the middle meatus beneath the middle turbinate, hence it is readily seen that any obstruction, such as enlarged middle turbinate, deviated septum, polyps, foreign objects, etc., to the drainage and ventilation of the sinuses will favor the development or prolongation of a sinus disease.

Group II is composed of the posterior ethmoidal and the sphenoidal sinuses. They drain into the superior meatus and likewise an obstructive lesion situated within the posterior portion of the superior meatus will favor the development or prolongation of a disease within these sinuses.

Pus emerging from beneath the anterior portion of the middle turbinate may be assumed to have its origin from one or more of the sinuses of Group I. Pus appearing above the posterior portion of the middle turbinate may be assumed to have its origin from one or both of the sinuses of Group II. The lacrimal duct and sac may be considered as an accessory sinus, but as it is described in the section on the eye it will not be considered here.

Etiology.—The most important predisposing cause of inflammatory diseases of the sinuses consists of an inadequacy of the drainage or ventilation of the sinuses, due to the various obstructive lesions of the nose.

The exciting causes are the microörganisms incident to the various infectious diseases.

Caries of the root of the second bicuspid and first molar, which sometimes perforate into the maxillary antrum, may produce an empyema of the antrum of a most virulent type.

The granulomatous or ulcerative processes of syphilis and tuberculosis sometimes invade the sinuses and give rise to inflammatory symptoms.

Pathology.—The changes which occur in the mucous membrane of the sinuses during a sinusitis are those incident to an inflammation of any mucous membrane, namely, congestion and small-celled infiltration with serous exudate mixed with mucus, bacteria and epithelial debris. The secretions, at first thin and watery, later become thick and tenacious. In many cases the inflammatory process passes from the catarrhal to the purulent type. As the catarrhal or suppurative disease becomes chronic many changes occur in the bone and mucous membrane of the sinuses. The membrane may have a granular appearance. Polyps may develop in any of the sinuses, although most frequently observed in the antrum and ethmoid cells. The membrane may be ulcerated in spots, exposing the underlying bone, which frequently is carious.

Symptoms and Diagnosis.—The subjective symptoms of an acute sinusitis, whether in the catarrhal or suppurative stage, are:

(*a*) Pain and headache usually referred to the region of the sinus involved, although in many cases this is not true, as for instance, the pain of an acute sphenoiditis or antritis may be referred to the frontal region, or the pain from an acute frontal sinusitis may be referred to the occipital region. The pain and headache may be dull, heavy and constant or sharp and shooting. The pain is usually increased when the patient leans forward.

(*b*) A sense of pressure or fulness between the eyes is characteristic of a frontal or ethmoidal infection. The patient may complain of the same feeling in the cheek or between the eyes when the antrum is involved.

(*c*) Giddiness and vertigo are sometimes present.

(*d*) Loss of smell due to the closure of the olfactory fissure is frequently complained of.

(*e*) Disturbances of the eye such as photophobia, epiphora, errors of refraction or accommodation, etc., may accompany the sinus disease.

The objective symptoms of an acute sinusitis are:

(*a*) Tenderness upon pressure over the affected sinus. This is nearly always present in the acute stage. In testing the frontal sinus the pressure should be exerted on the floor of the sinus near the inner angle of the orbit, and over the anterior wall above the supra-orbital ridge. The anterior ethmoid cells should show tenderness over the orbital plate of the ethmoid at the inner angle of the orbit. In examining the antrum pressure should be made over the canine fossa of the superior maxilla.

(*b*) A mucopurulent or a purulent secretion may be observed in many cases as it emerges from beneath the tip of the middle turbinate or from above the middle turbinate, depending upon whether the sinuses of Group I or Group II are affected.

(*c*) Transillumination of the face is a valuable aid in diagnosing an empyema of the antrum. Three points should be noted with an infected antrum, namely: (1) The luminous pupil is dim or absent. (2) The intra-orbital crescent of light is dim or absent. (3) When the patient's eyes are closed he does not perceive a sense of light in the eye above the affected antrum.

Transillumination is of doubtful value in diagnosing a frontal sinusitis and of no value in the sinuses other than the maxillary and frontal.

(*d*) Irrigation (see page 359) of the affected sinus is frequently resorted to, to determine the presence or absence of pus.

The symptoms of chronic suppurative sinusitis.

The chief symptoms of a chronic empyema of a sinus are the discharge of pus from the nose and the constant or occasional headache, together with the various manifestations in other portions of the body that may accompany any focal infection.

The pus or mucopurulent secretion is usually continuous, although more noticeable in the morning. Frequently no secretion is noticed during the day.

The pain and headache depends upon the degree of retention. In the chronic form partial or complete retention does not occur as frequently as in the acute stages. In frontal sinus empyema the pain over the temple and eye may resemble a neuralgia. A maxillary empyema may also give a frontal headache, usually unilateral unless sinuses on both sides of the head are involved.

Chills and fever are rarely present in a chronic sinusitis. They are frequently present in the first stage of an acute sinusitis.

The *x*-rays are a most valuable aid in diagnosing the presence of a diseased sinus. In addition the *x*-rays reveal the presence or absence, the location, and the dimensions of the various sinuses, a consideration of great value when surgical procedures are contemplated.

If the sinus is diseased or pus filled, its outline on the negative is not clear and distinct and its area is cloudy or hazy. A healthy sinus should appear clear and dark. A polypoid condition is suggested by a mottled appearance; while a thickened or inflamed mucous membrane presents a uniform shadow.

A complete examination is justifiable in most cases and should consist of (1) postero-anterior; (2) lateral (preferably stereoscopic); (3) two obliques (Figs. 119 and 120). Where the expense must be considered, one postero-anterior and one lateral will give the most information. To obtain a postero-anterior view M. J. Hubeny advises that the patient be placed prone with the forehead and nose pressed firmly on the plate. The tube is placed about twenty inches from the plate in such a position that the central beams pass through the median line and in a postero-anterior plane, which extends from the base of the nose through the external auditory meatus. In reading such a plate from above downward, one will see: (1) frontal sinuses; (2) cribriform plate and a few ethmoid cells; (3) sphenoid sinuses; (4) posterior ethmoid cells, located laterally to the sphenoids, and just below the posterior group of ethmoid cells are the anterior ethmoid cells; (5) nasal cavity with the septum and turbinates; (6) maxillary sinuses just below the orbits.

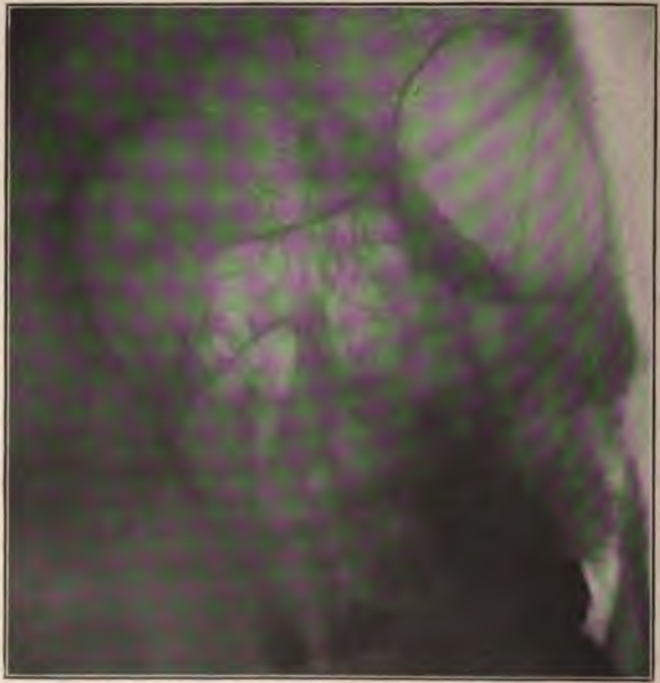

FIG. 119.—Right oblique view of normal sinuses.

In the lateral view the tube is adjusted so that the central beam will bisect a line connecting the external auditory meatus and the external orbital margin and directed perpendicularly to the plate. This plate will show: (1) external auditory meatus; (2) sphenoid sinuses; (3) sella turcica; (4) posterior and anterior ethmoid cells; (5) frontal sinuses; (6) orbit; (7) maxillary sinuses.

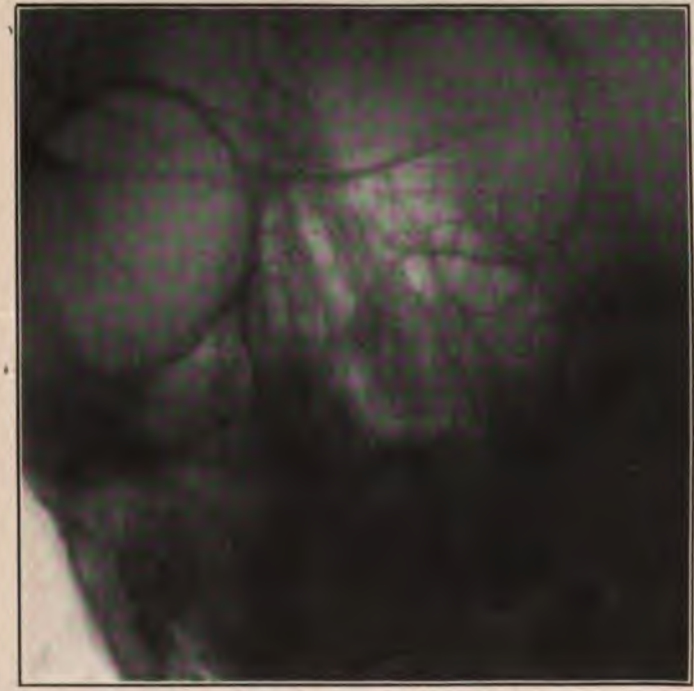

FIG. 120.—Left oblique view of normal sinuses.

In the oblique position (Fig. 121) the superciliary ridge, the nose, and the malar bone should touch the plate. Both sides should be taken for comparison. The central beam should enter the opposite parietal region about two inches posteriorly and one and one-half inches above the external auditory meatus and projected toward the centre of the orbit. The optic foramen and the sphenoidal sinus will be seen near the centre of the orbit. Above the orbit the frontal sinus and below the orbit the maxillary sinus will be found. The ethmoid cells will be seen extending from the sphenoidal sinus to near the bridge of the nose. By this method the writer has been able to determine a localized ethmoiditis, where only one or two cells were apparently involved.

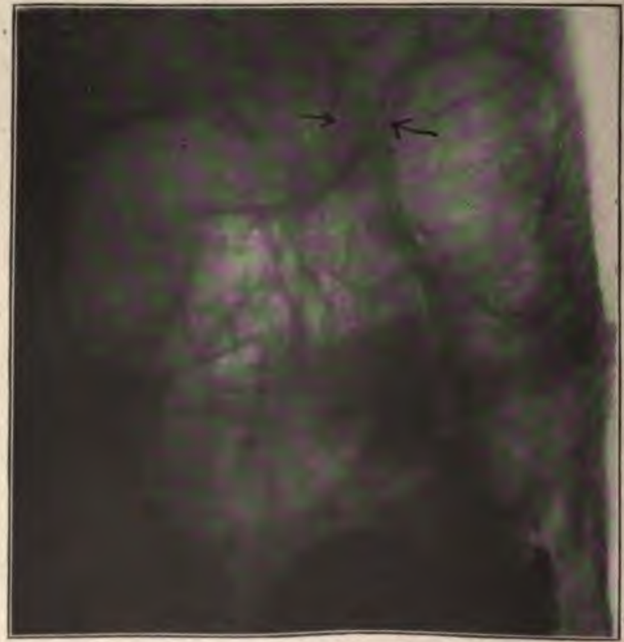

Fig. 121.—Right oblique view of the diseased bulla frontalis as shown in Fig. 122.

Treatment.—The treatment of sinus disease includes: (*a*) the removal of any obstructive lesions; (*b*) the establishment of adequate drainage and ventilation and (*c*) the removal of the morbid tissue.

In the acute catarrhal or acute suppurative stage of sinus disease the establishment of adequate drainage and ventilation is usually sufficient to effect a cure. This is probably best and most easily accomplished by the local application of a 2 to 5 per cent. cocain or alypin solution, combined with a 1 to 10,000 adrenalin chlorid solution to the swollen and inflamed mucous membrane. When any of the sinuses in Group I are affected, care should be taken to reduce the congestion in the region of the infundibulum beneath the middle turbinate.

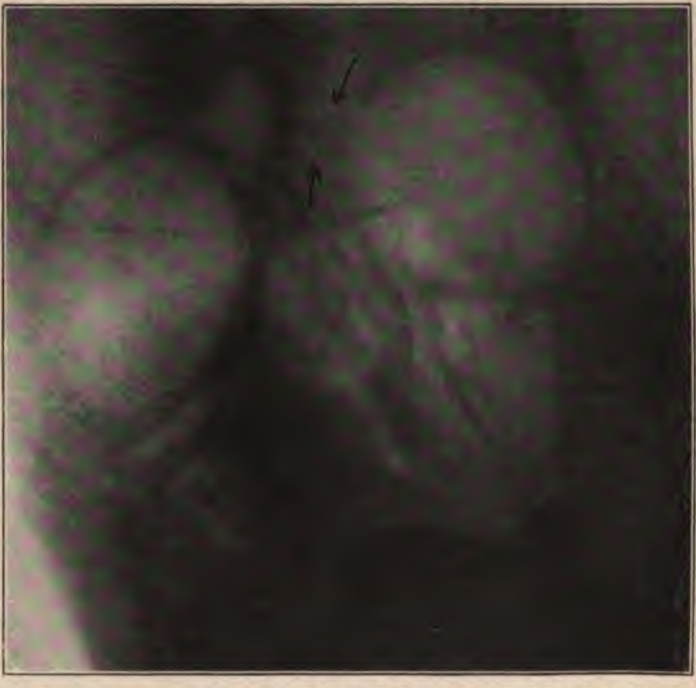

FIG. 122.—Involvement of the bulla frontalis on the left side. The frontal sinus is normal. (See Fig. 121.)

The removal of the secretions may be greatly facilitated by the use of a suction apparatus, or by daily irrigations of the affected sinuses with a warm saline or boric-acid solution.

Irrigation of the frontal sinus is accomplished by inserting a slender, silver, frontal sinus cannula, bent into the proper shape, through the frontonasal duct, attaching a syringe to the cannula and irrigating with the warm solution. The cannula should be bent at its distal end to an angle of about 135 degrees. When inserting the cannula or probe, the tip should be passed along the outer surface of the turbinate forward and upward through the hiatus into the infundibulum where in most cases it readily enters the frontonasal canal. Irrigation of the maxillary sinus may be done through the normal ostium, by means of a Pierce antrum cannula or through an artificial opening made beneath the inferior turbinate by means of a trocar. The technic of the latter method is as follows: (*a*) anesthetize the mucous membrane of the outer wall of the inferior meatus; (*b*) introduce the trocar and cannula beneath the inferior turbinate about one inch posterior to the inferior border of the nostril and slightly above the floor of the nose; (*c*) penetrate the naso-antral wall in an upward and outward direction; (*d*) remove the trocar, leaving the cannula in position; (*e*) attach the syringe to the cannula and irrigate. During the irrigation the patient's head should be bent forward over a pus basin.

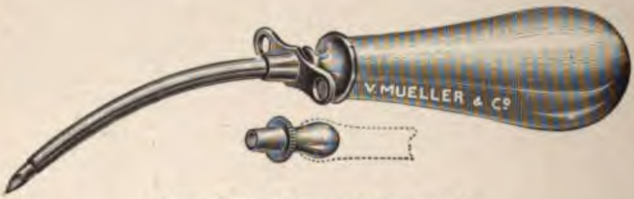

FIG. 123.—Kyle's antrum trocar.

If the irrigations are to be continued for some time and the opening persists in closing, it may be enlarged by means of a burr, chisel, or punch forceps. Irrigation of the sphenoid sinus is possible where an enlarged middle turbinate or a deflected septum does not prevent the introduction of the sphenoid cannula. When such an obstruction is present, it may be necessary to remove it before the irrigations can be satisfactorily practised.

Irrigation of the ethmoid cells is often impossible except in the case of the two or three anterior cells which drain into the frontonasal duct.

The instillation of a 10 to 25 per cent. argyrol solution into the infundibulum or sinuses is often followed by good results.

When the frontal sinus is the seat of an acute inflammatory trouble, cold or hot applications are very agreeable.

Administration of Autogenous Bacterial Vaccines.—Good results are frequently obtained from the administration of the bacterial vaccines. The autogenous vaccine will produce the following results.

Two or three minims of vaccine, each minim containing 100,000,000 killed virulent bacteria, suspended in 0.5 per cent. carbolic acid, will, if injected strictly subcutaneously, produce a distinctly inflamed painful areola about two inches in diameter. This is determined by the virulence of the bacteria injected. Use a sterilized syringe and inject into a sterilized field of operation.

The areola disappears within forty-eight hours without suppuration. Two or three days after the disappearance of the areola another injection should be made. Later on it is necessary to increase the dose to produce the same sized areola. Decreasing areolæ after increasing doses of vaccine are due to beginning acquired immunity. After the bacteria in the lesion have disappeared, injections should be continued once a week for at least a month to keep up immunity and thereby prevent relapse. The largest dose should not exceed 10 minims or 1,000,000,000 killed bacteria.

If the above measures, together with the removal of any intranasal obstructive lesions, do not effect a cure of the affected sinuses after a thorough trial, it will be necessary to resort to some surgical procedure.

SURGERY OF THE ACCESSORY SINUSES.

The various operations upon the nasal sinuses may be divided into the intranasal and the external method. It may be stated that the external operations should be resorted to only as a last resort, when the intranasal methods have failed or would probably fail.

The Frontal and Ethmoidal Sinuses.—Intranasal Operations. —Probably the most simple intranasal operation on these sinuses is the removal of the anterior tip of the middle turbinate; however, as this operation is described elsewhere, it will not be considered here.

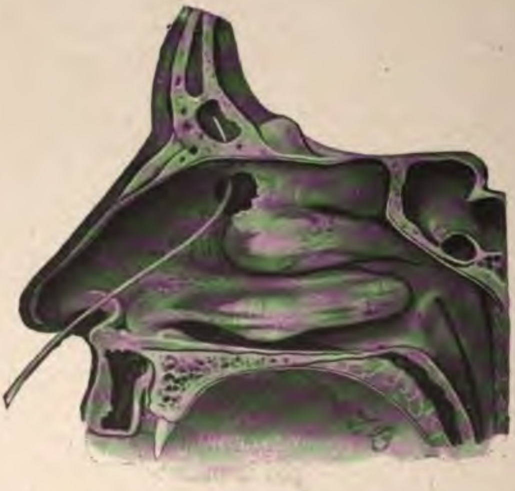

Fig. 124.—Mosher's fronto-ethmoid operation. Entrance into the frontal sinus has been obtained.

Mosher's Fronto-ethmoid Operation.—This operation is preferably performed under local anesthesia.

An ethmoid curette is introduced with its cutting edge toward the orbit until the tip reaches a point just above the anterior attachment of the middle turbinate. The thin plate of bone at this point is broken through (Fig. 124) and a portion of the anterior ethmoid cells are broken down. The curettage is continued in an upward and forward direction until the frontonasal opening in the floor of the

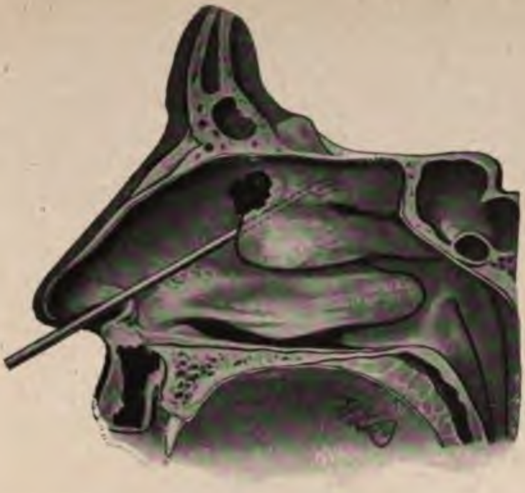

FIG. 125.—Mosher's fronto-ethmoid operation. The removal of the remaining anterior and posterior ethmoid cells.

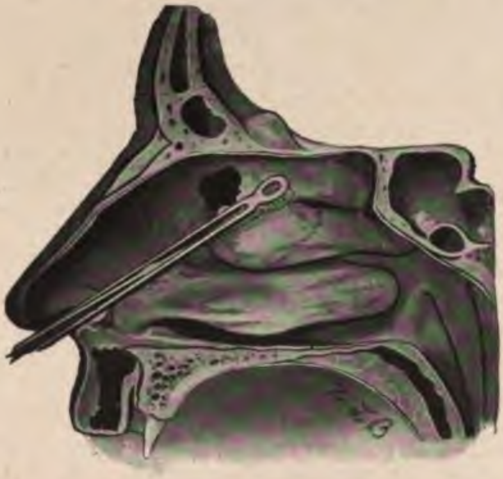

FIG. 126.—Mosher's fronto-ethmoid operation. The turbinal plate may be removed by grasping with suitable dressing forceps.

frontal sinus is enlarged and free drainage established. The next step is the removal of the remaining anterior and posterior ethmoid cells. This is done by inserting the curette through the opening made into the turbinal plate and breaking down the cells between this plate and the orbital plate until the anterior wall of the sphenoid sinus is reached. The turbinal plate, consisting of the superior and middle turbinate may now be removed or left intact. If removed, it is grasped with suitable dressing forceps and with a careful twisting motion is fractured from its attachments and removed. No dressing is necessary unless bleeding is profuse. If a loose packing is made, it should be removed in twenty-four hours.

Watson Williams uses a small angular ethmoid forceps to engage the anterior margin of the middle turbinate at its point of attachment to the outer nasal wall. Cutting through this the forceps enter the anterior ethmoidal cells in front of the frontonasal duct. All the nasi-agger cells and other cells are removed by the forceps up to the nasal crest and as far back as desired. After exenterating the ethmoid cells and establishing as large an opening as possible through the floor of the frontal sinus the anterior tip of the middle turbinate is removed to create free drainage.

Good's Operation.—Good first removes the anterior tip of the middle turbinate, then introduces a guard or guide into the frontonasal canal. A rasp, the file edge facing anteriorly, is then pushed along the course of the frontonasal canal until the tip enters the frontal sinus. The anterior ethmoid cells are broken down as the rasp is inserted.

Halle's Operation.—Halle uses a burr to gain entrance to the frontal sinus by the intranasal route. The internal plate of the frontal bone is guarded against injury by a grooved protector.

The Removal of the Ethmoid Cells and Middle Turbinate En Masse.—This may be accomplished by means of the Ballenger ethmoturbinate knives. The principal knife consists of a short blade set at right angles to a longer blade, which is parallel to and a continuation of the shank of the instrument.

Technic.—1. Local anesthesia is preferable, although general anesthesia may be given.

2. The right-angle knife is introduced into the middle meatus with the short blade turned upward (position *a*, Fig. 127) until it comes in contact with the sphenoid bone.

3. The short blade is then pushed outward, severing the posterior ethmoid cells from their attachment to the body of the sphenoid.

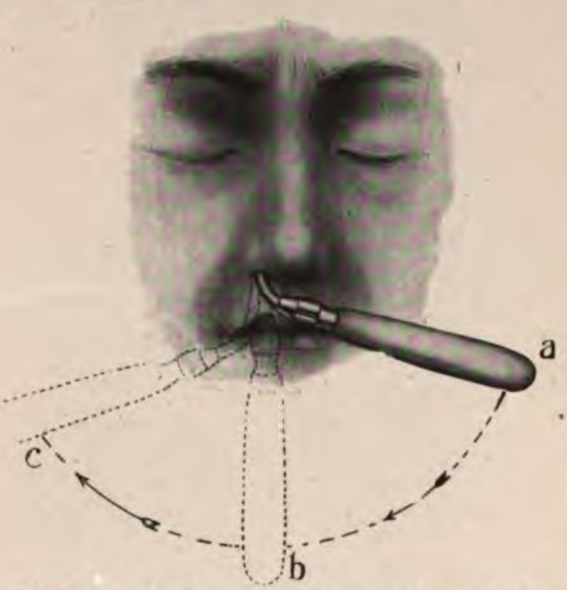

FIG. 127.—Showing the three positions (*a*, *b*, *c*) of the ethmoid knife, in the successive steps of Ballenger's exenteration of the middle turbinate and ethmoid cells *en masse*.

4. The handle of the knife is then rotated about 90 degrees to position *b*, Fig. 127.

5. The short blade is now in a parallel position to the cranial plate and the long blade is parallel to the orbital plate (Fig. 128). The instrument is now drawn forward with a slight oscillating movement to the anterior attachment of the middle turbinate.

6. As the anterior portion of the superior meatus is quite narrow, the handle of the knife is rotated another arc of about 90 degrees, as shown in position *c*, Fig. 127. This movement brings the long blade of the knife on top of the detached mass and the short blade downward into the nasal chamber.

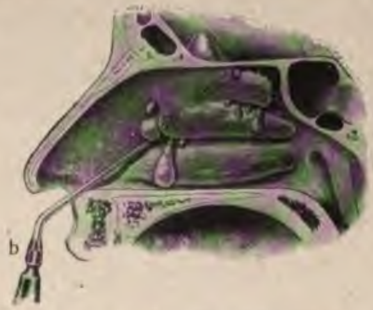

Fig. 128.—The second step of the ethmoidal operation. The instrument in position *b*, Fig. 127.

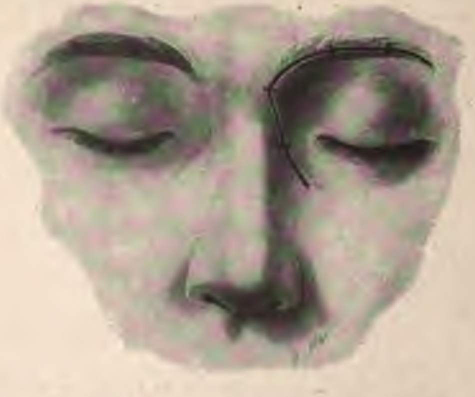

Fig. 129.—Killian's incision, with cross-cuts for guides in suturing.

7. The knife is now drawn forward and downward to complete the severance of the tissues.

8. If the mass is not delivered with the withdrawal of the knife, it may be seized with a dressing forceps and removed. If the specimen is still attached to the nasal walls by shreds of tissue, the blunt hook knife should be introduced and the attachments severed.

9. No dressing or packing is advisable unless excessive bleeding occurs. If a packing is used, it should be removed in from one to twenty-four hours.

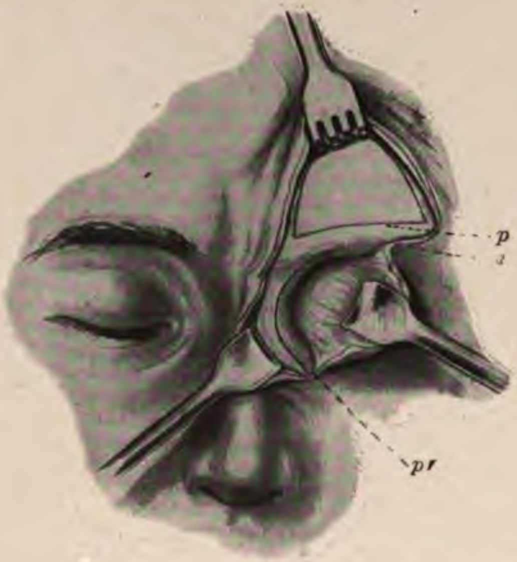

Fig. 130.—Showing the retraction of the skin flaps in the Killian frontal sinus operation: *p*, the periosteal incision 5 mm. above the skin incision; *s*, the skin incision 5 mm. below the periosteal incision; *p′*, the periosteal incision at the side of the nose.

External Operations.—*Killian's Operation.*—This is probably the most thorough and consequently the best external operation on the frontal and ethmoid sinuses. A general anesthetic should be given. An incision, including the skin and muscle, but not the periosteum, is made through the eyebrow beginning at its temporal end and extending to the root of the nose, and then curved downward and outward below the base of the nasal bone (Fig. 129). The incision may be marked with small cross-cuts to aid in perfect coaptation at the finish of the operation.

The periosteum is incised in two places. The upper incision is made parallel with and about 5 mm. above the supra-orbital ridge from the outer extremity of the wound to the root of the nose. The second incision is made from a point below the supra-orbital ridge and internal to the attachment to the pully of the superior oblique muscle. It passes inward and downward and then curves outward, following the skin incision (Fig. 130). This leaves the periosteum intact over the superciliary ridge. The dislodgement of the pully of the superior oblique muscle is thus largely prevented.

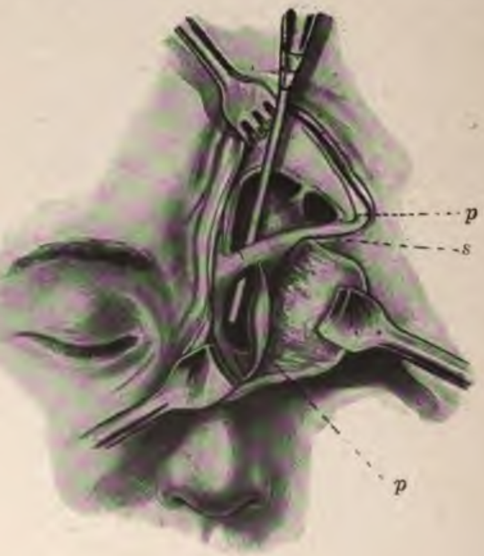

FIG. 131.—The Killian frontal sinus operation completed: *p*, the periosteal incision 5 mm. above the superciliary skin incision; *s*, the superciliary skin incision 5 mm. below the periosteal incision; *p′*, the periosteum elevated and everted along the side of the nose.

Having elevated the soft parts, including the periosteum, the frontal sinus is entered by means of a trephine or gouge and the entire anterior wall of the sinus removed with a gouge and rongeur forceps.

The sinus is thoroughly curetted of its contents, including its mucosa and all septa are removed. The next step is the removal of the floor of the sinus. A curette may be used for this purpose. Care should be taken to avoid injuring the bridge of bone.

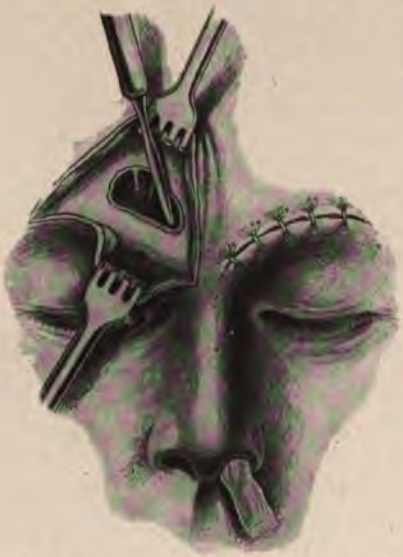

FIG. 132.—The Hajek-Luc operation. The anterior wall of the frontal sinus is removed, and the anterior ethmoidal cells are being removed through the floor of the frontal sinus with a curette. The left side has been operated on, a gauze wick introduced through the anterior ethmoidal wound and drawn out through the nostril.

The frontal process of the superior maxillary bone and a portion of the nasal bone are removed by means of a chisel to insure a more easy and thorough removal of the ethmoid cells. The removal of the floor of the sinus should unite the two external openings as shown in Fig. 131. A gauze drain should be inserted from the frontal sinus into the nasal cavity and the periosteum and skin united with sutures.

Kuhnt's Operation.—Kuhnt resects the anterior wall and a portion of the floor of the frontal sinus. He curettes the sinus thoroughly, but does not disturb the frontonasal canal. Drainage is established through the external wound.

Hajek-Luc Operation.—The anterior wall of the frontal sinus is removed, and the anterior ethmoid cells are removed through the floor of the frontal sinus. The frontonasal canal is enlarged and a rubber tube is inserted.

The Maxillary Sinus.—Intranasal Operations.—The simplest intranasal operation is the puncture with a trocar of the naso-antral wall beneath the inferior turbinate as described under Irrigation of the Maxillary Sinus. As the small opening tends to close quickly it is sometimes desirable to make a larger opening.

Removal of the Naso-antral Wall.—Vail first removes the anterior half of the inferior turbinate by means of a knife or scissors, then punctures the naso-antral wall near the floor of the nose. Through this opening the Vail antrum saw, which is lightly curved on the flat, is introduced in an oblique direction and a circular section of the wall is incised and removed. The dressing consists of iodoform gauze loosely packed into the sinus. The gauze is removed in from twenty-four to forty-eight hours.

Fletcher has devised an antrum punch which is a very useful instrument. It is made in two forms, the original heavy right-angle cutting instrument and one curved instrument adapted to the universal handle. Fletcher recommends the original instrument as the better, although the latter is less bulky. The advantage of these instruments is that they cut in any direction without change of position of the hand.

Corwin has devised an antrum chisel with projecting points which facilitate the engagement of the chisel at an acute angle in the antral wall.

Stein's Operation. Stein uses a hand gouge with a "V" shaped cutting edge, one point of which he enters through the naso-antral wall below the inferior turbinate. The gouge is pushed backward, keeping the cutting edge as high as possible, until the incision is the desired length. The instrument is withdrawn and reintroduced at the point the first incision was begun, only directing the cutting edge of the instrument as close to the floor of the meatus as possible and driving backward and finally upward to meet the posterior end of the first incision.

If desired, a portion of the inferior turbinate may be removed with the naso-antral wall by making the first incision above the inferior turbinate and extending the incision downward through the inferior turbinate at the selected point.

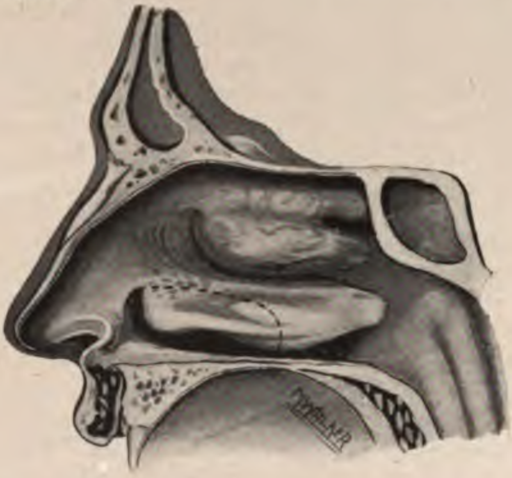

FIG. 133.—The dotted line shows the portion of the naso-antral wall to be removed in the intranasal antrum operation. A portion of the inferior turbinate is usually removed.

Canfield-Ballenger Antrum Operation.—This is an intranasal operation designed to preserve the inferior turbinate intact. The operation consists in the removal of that portion of the naso-antral wall lying beneath the attachment of the inferior turbinate and the portion lying anteriorly to the anterior tip of the inferior turbinate, including the anterior angle of the antrum.

The mucous membrane is anesthetized by the local application of cocain. The skin over the canine fossa is anesthetized by the subcutaneous injection of a 4 per cent. solution of novocain. This may be done through the vestibule of the nose.

An incision is made through the skin of the vestibule at the margin of the pyriform aperture. The soft tissues, including the periosteum, is elevated over the canine fossa. Entrance is gained into the antrum through the margo-pyriformis by means of a gouge and mallet or a rongeur bone forceps. As much of the canine wall may be removed as will fully expose the antrum. The naso-antral wall is next removed by placing the Wagner forceps astride the partition and biting it away from the attachment of the inferior turbinate to the floor of the nose. Curettage of the mucous membrane of the antrum should not be done unless excessive polyps or granulation tissue is present. A gauze drain should be placed in the antrum through the naso-antral opening and removed the following day. The antrum should be cleansed daily until the purulent discharge stops.

External Operations.—*Alveolar Method.*—The removal of a tooth, usually the second bicuspid or first molar, to establish drainage from the antrum is, obviously, only advisable when there is positive evidence that the tooth is diseased beyond hope of repair.

Caldwell-Luc Operation.—This operation consists in the removal of the anterior wall of the antrum and a section of the naso-antral wall. The upper lip is elevated and an incision is made at the labiogingival junction through the soft tissues, including the periosteum. The periosteum is elevated over the canine fossa and the anterior wall of the antrum is removed by means of a chisel and rongeur forceps. The anterior two-thirds of the inferior turbinate and the naso-antral wall are removed as described under Intranasal Operations. All diseased tissue such as polyps and granulations should be removed from the antrum cavity and a strip of gauze inserted, one end of which is brought out through the nose. The labiogingival incision is closed, as the drainage through the naso-antral opening is usually sufficient.

Denker's Operation.—A general anesthetic is usually given. A labiogingival incision is made as in the Caldwell-Luc operation, but extends to the median line of the nose. In addition to removing the anterior wall of the nose, the bridge of bone left between the canine fossa and the pyriform aperture is removed, thus obliterating the anterior angle of the antrum. The mucoperiosteum covering the inferior turbinate and the floor of the nose is elevated. The naso-antral wall, including the denuded portion of the inferior turbinate is removed and the mucoperiosteal flap is turned outward on the floor of the antrum and held in place for twenty-four to forty-eight hours with loosely packed gauze.

Daily cleansing and the control of exuberant granulations comprise the after-treatment.

The Sphenoid Sinus.—The operation on the sphenoid sinus consists of (1) a preliminary removal of the middle turbinate, to expose the sphenoidal ostium to view and (2) the removal of the anterior wall of the sphenoid. Fletcher's sphenoidal punch is admirably adapted for this purpose, as it cuts in all directions. It may be necessary to enlarge the sphenoidal ostium with a curette before the forceps can be introduced, although the conical projection on the distal end of the Fletcher punch enables the operator to force the instrument through the natural opening in most cases.

NEOPLASMS.

Myxomata (*Polypi*).—Myxomata have already been considered on page 327.

Papillomata.—**Synonyms.**—Warty or Cauliflower-like Tumors.

Definition.—These are benign growths which appear at the anterior margin of the nares, at the junction of the skin and mucous membrane, or upon the mucous membrane itself. They are usually unilateral.

Treatment.—As the tumor is benign, it need not be removed unless it is the source of obstruction or irritation to the respiratory tract. If, however, their presence is a source of obstruction and irritation, they should be removed after thorough cocainization of the parts. A knife, the cold-wire snare, or cautery may be used for this purpose. As the growth is more or less pedunculated, the operation is easily performed. If the knife or snare is used, the wound should be cauterized to prevent recurrence of the growth.

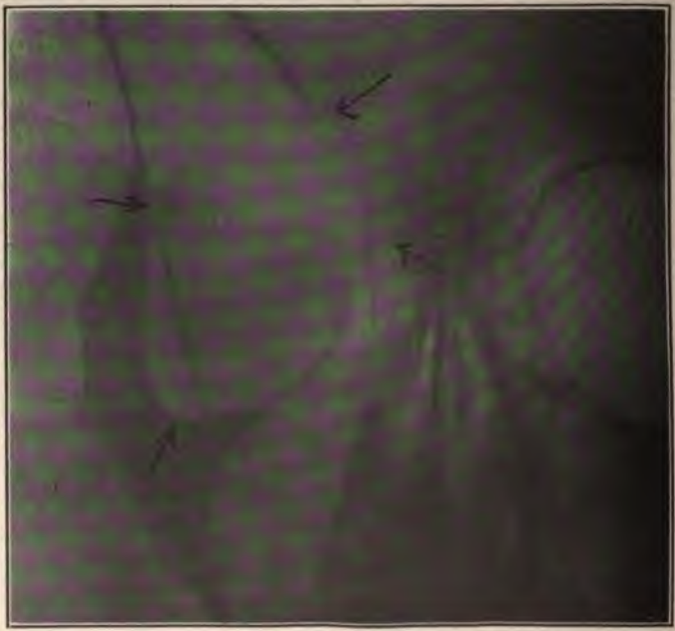

FIG. 134.—Cyst of the frontal bone, involving the frontal sinus.

Adenoma.—This type of tumor in the nares is rare, as there is but little glandular structure in the mucosa of the nose. When simple, it is benign, but it more often occurs in mixed tumors of the cancerous type. When it occurs as a simple adenoma, it is composed of true glandular tissue, and not of lymphoid tissue, of which there is an abundance in the nasal mucosa.

Cystoma of the Nasal Passages.—**Definition.**—This is a cystic formation of the nasal mucosa from degeneration of a lymphoid nodule; or from closure of the mouth of a mucous gland. When formed from a lymphoid nodule, it is the result of a degeneration by which the contents of the mass are liquefied, and a limiting membrane is formed from inflammatory reaction at the periphery. The appearance is like that of a polypus, except as to the fluid contents.

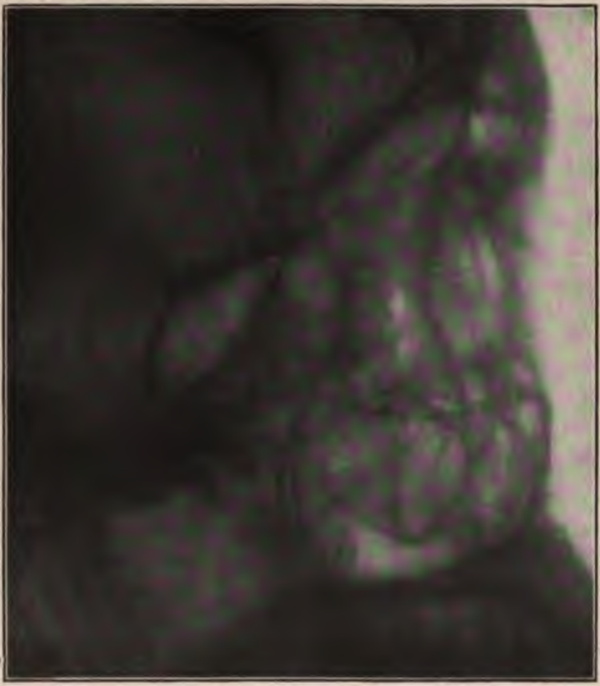

FIG. 135.—Lateral view of cyst of frontal bone as shown in Fig. 134.

Treatment.—The treatment is that of polypi.

Angiomata of the Nasal Passages.—This condition is rare, which is rather remarkable in view of the fact that the nasal mucosa is very vascular.

Etiology.—Angiomata are due to nutritive changes in the walls of the bloodvessels, rather than to changes in the mucosa.

Symptoms.—Epistaxis occurs frequently. The presence of the tumor excites considerable irritation, as is shown by the mucopurulent discharge. The nose does not usually show external deformity. The tumors are red or purplish, according to the predominance of arterial or venous blood within them. They are irregularly nodular, and often bleed profusely from slight wounds inflicted during examination.

Prognosis.—Their development covers a period of from two to five years. Life is not usually greatly endangered, although hemorrhage may at times be alarming or even fatal.

Treatment.—Their removal is not followed by recurrence. The cold snare adjusted at the base of the tumor and very gradually tightened affords the safest means of operation. An hour or more should be used for this purpose, as haste might be attended by serious hemorrhage. The application of a freshly prepared solution of adrenalin chlorid immediately after cocainization of the parts will contract the small arterioles and greatly diminish the amount of hemorrhage.

Chondroma of the Nasal Passages.—Occurrence.—This form or tumor is rarely seen in the nasal passages. The few cases reported have occurred chiefly during the period of adolescence. The growth is composed of hyaline cartilage and may vary greatly in size.

Symptoms.—Upon inspection it appears as a round, hard tumor, and very much resembles a fibroma. When of considerable size, it gives rise to stenosis. Chondromata are usually located at the junction of the cartilaginous septum with the anterior nasal cartilages. They are benign and give rise to no serious symptoms beyond those due to nasal obstruction.

Treatment.—They should be removed when thus offending.

Fibroma of the Nasal Passages.—The etiology is obscure. Inflammation and disturbed nutrition of the parts seem to favor development. About two-thirds of the cases occur in males. While the tumor may occur at any age, it is most often found between the ages of fifteen and thirty.

Symptoms.—The symptoms are those peculiar to nasal obstruction. In appearance the growth is smooth, hard, and rounded in contour, and is covered with healthy mucous membrane. Epistaxis is a prominent symptom, and may be so severe as to render the prognosis grave. It invades any kind of tissue, and hence may grow in any direction.

Prognosis.—The prognosis is grave unless the neoplasm is thoroughly removed. Its rapid, relentless growth through the adjacent tissues renders it a serious menace to life, as it may invade the cranial cavity and other important structures.

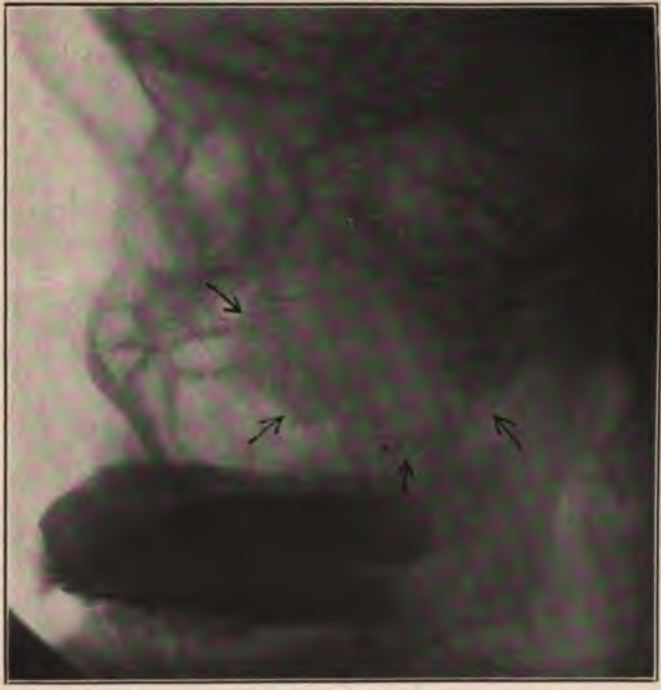

FIG. 136.—Sarcoma of the antrum and epipharynx. (Author's case.)

Treatment.—The treatment is surgical, the snare perhaps affording the best means for its removal. It may be necessary to do the operation under a general anesthetic, turning the lip and nose of the patient upward over the brows so as to expose more fully the nasal cavities. The danger attending the operation is from hemorrhage.

In other cases it may be necessary to make a temporary resection of the superior maxilla to fully expose the growth.

Osteoma of the Nasal Passages.—Definition and Occurrence.—This is a form of bony neoplasm which usually grows from the accessory cavities or the upper part of the nose. It involves other tissues lying in its path, and often causes marked facial deformity. Males are more frequently affected than females, and it is more common before middle life.

Symptoms.—External deformity, nasal stenosis, and pain are the prominent symptoms.

Treatment.—A radical external operation is usually required for its removal, after which it shows but slight tendency to recur.

Sarcoma and Carcinoma of the Nasal Passages.—Sarcoma.—It usually *occurs* about middle life or at a much earlier period. Sarcoma of the nose runs a slower course, and is not so malignant as sarcoma in other parts of the body.

The prominent *symptoms* are nasal obstruction, repeated attacks of epistaxis, and pain.

Treatment in most cases consists in its removal by a radical external operation.

Carcinoma.—Primary carcinoma is rare in this locality.

It usually occurs after the fortieth year. Early diagnosis and complete removal offer the only hopes of cure.

THE PHARYNX.

SURGICAL ANATOMY OF THE PHARYNX.

The pharynx is divided into the *nasopharynx*, or that portion of the pharynx situated above the soft palate, the *oropharynx*, the portion between the soft palate and the hyoid bone and the *laryngopharynx* which extends from the hyoid bone to the lower border of the cricoid cartilage.

Nasopharynx.—The nasopharynx is formed above by the body of the sphenoid and the basilar process of the occipital bone, anteriorly by the choanæ and the soft palate, posteriorly by the cervical vertebræ, and inferiorly the nasopharynx is continuous with the oropharynx. Situated on the lateral walls of the nasopharynx, behind the posterior ends of the inferior turbinates are the orifices of the Eustachian tubes. Above and behind the Eustachian orifice is an elevation or ridge, formed by the Eustachian cartilage, called the Eustachian cushion. Extending downward from the posterior end of the ridge is a strong fold of mucous membrane, the salpingopharyngeal. A less prominent fold of mucous membrane, the salpingopalatine, extends downward in front of the Eustachian orifice. The deep pocket formed at the angle of the pharynx between the posterior ridge of the Eustachian cartilage and the posterior wall is known as the fossa of Rosenmüller. Frequently adenoid tissue is found around the orifice of the tube (tubal tonsil). The roof (fornix pharyngis) and the posterior wall of the nasopharynx are the seat of lymphoid tissue (adenoid, pharyngeal tonsil, Luschka's tonsil) which frequently attains considerable size, especially in children.

The pharyngeal tonsil or adenoid is a lobulated, lymphoid mass, whose lobules are frequently arranged in regular order around a central depression, sometimes called the bursa pharyngea. Many pockets and crevices are thus presented.

Oropharynx.—The oropharynx opens into the oral cavity at the anterior pillar of the fauces.

The soft palate (velum palati) consists of muscle fibers supported by a fibrous tissue and an outer covering of mucous membrane. A median ridge or raphe divides it into two halves. The conical shaped central projection is known as the uvula. The lateral margins of the palate, on each side, divide into the anterior and posterior pillars of the fauces. The anterior pillar contains the palatoglossus muscle. The posterior pillar contains the palatopharyngeus muscle. Between the two pillars is the recess in which the faucial tonsil is lodged.

The faucial tonsils, one on each side of the oropharynx, are almond-shaped masses of lymphoid tissue imbedded in a fibrous capsule. The inner or free surface is covered by a closely adherent epithelial membrane. This epithelium extends into the blind pouches or crypts which have their openings onto the surface of the tonsil. These crypts may be very shallow or they may extend the entire depth of the tonsil.

The plica triangularis is a thin fold of mucous membrane, stretching backward from the anterior pillar and covering a portion of the anterior surface of the tonsil.

The supratonsillar fossa is a recess of variable size situated above the tonsil and between the anterior and posterior pillar. It is formed embryologically from the second branchial cleft.

The lingual tonsil is a mass of lymphoid tissue situated on the base of the tongue. It is divided into two halves by the median glosso-epiglottic ligament.

The pharyngeal, faucial and lingual tonsils form the so-called Waldeyer's ring.

Laryngopharynx.—The laryngopharynx is partially separated from the oropharynx by the pharyngo-epiglottic fold, which extends from the epiglottis to the side of the pharynx.

The mucous membrane of the pharynx is covered with squamous epithelium. Many mucous glands are present. It also contains much lymphoid tissue. This lymphoid tissue is collected into small masses (lymphoid follicles) at numerous points throughout the pharynx.

The muscles of the pharynx consist of the three constrictors of the pharynx, the superior, middle and the inferior, the stylopharyngeus and the palatopharyngeus.

The muscles of the soft palate are the azygos uvulæ, levator palati, tensor palati, palatoglossus and palatopharyngeus.

The blood supply from the pharynx comes from many sources and is sometimes irregular. The chief supply is derived from the ascending pharyngeal and faucial branches of the external carotid and from the superior palatine of the internal maxillary. The principal supply to the faucial tonsil is the tonsillar branch of the facial artery.

DISEASES OF THE PHARYNX AND FAUCES.

Acute Pharyngitis.—**Etiology.**—The predisposing causes are: (1) Lowered vitality from unhygienic surroundings. (2) Constitutional disorders, digestive, rheumatic, etc. (3) Exposure to cold or dampness. The exciting cause is usually an infection. The pneumococcus, streptococcus, and the influenza bacillus probably being most frequently found. An acute pharyngitis is nearly always present in such diseases as measles, scarlet fever, smallpox, etc. Irritants such as dust, chemicals, etc., may produce an acute pharyngitis. The pharyngitis may be secondary to an acute or chronic rhinitis or tonsillitis.

Symptoms.—The functional symptoms are dryness of the throat, painful deglutition, hawking or hemming. The *objective symptoms* embrace a dry, red, swollen membrane, which later is covered with a thick, tenacious mucus. This at a still later period may become mucopurulent. The *constitutional symptoms* begin with fever (usually high in children), a dry coated tongue, constipation, indigestion, headache, and frequently muscular and joint pains over the whole body.

Treatment.—This should begin with the administration of calomel, followed by a brisk saline purgative. Aconite in one-drop doses may be given until the pulse goes down and the skin becomes moist.

The Local Treatment.—Lozenges of guaiacum, 2 grains each, are especially useful when there is a rheumatic diathesis. Menthol, $\frac{1}{4}$ grain, or menthol combined with oleum eucalyptol, forms a grateful and efficient paste or troche. Tannate of glycerin, steam inhalations, and applications of silver nitrate are of doubtful value in the acute stage. The sucking of ice or cold sprays are very grateful. When pain and great congestion are present equal parts of guaiacol and sweet oil, as a local application, are a most efficient remedy. Menthol, 20 parts, to 80 parts of olive oil, should be used in less severe cases. Scarification may become necessary when edema develops.

Retropharyngeal Abscess.—**Etiology.**—This is an accumulation of pus in the connective tissue beneath the posterior wall of the pharynx. Traumatisms, as from fish bones and other foreign bodies, may also give rise to it. Occasionally it may arise from nasal or middle-ear inflammation. The syphilitic and strumous dyscrasia predispose to it. The infectious diseases of childhood probably produce the larger proportions of all cases. Caries of the cervical vertebræ is an occasional cause.

Symptoms.—The subjective or functional symptoms may be classified as follows:

(*a*) The act of swallowing is interfered with.

(*b*) If the abscess is low down, dyspnea becomes an important symptom.

(*c*) The respiration is stertorous; and if the larynx is involved, it is stridulous.

(*d*) If the vertebræ are diseased, rotary movements of the head cause pain.

(*e*) The temperature may or may not be elevated.

Objectively, a fluctuating swelling may be seen upon the posterior wall of the pharynx. Aneurisms should be excluded before incision is made to evacuate the pus.

Prognosis.—Those cases associated with caries of the spine are grave. In children there is in all types of pharyngeal abscess the danger of strangulation from rupture during sleep.

Treatment.—As soon as a diagnosis has been made the abscess cavity should be freely lanced, the head thrown forward, and the pus expectorated. If the abscess is low down, it may be necessary to enlarge the opening with the index finger. Local anesthesia should be secured by applying cocain. The incision should be made in the median line, so as to avoid injuring the internal carotid.

The abscess should be reopened whenever the pus reaccumulates.

Chronic Pharyngitis. — **Synonyms.** — Clergyman's Sore Throat; Granular Pharyngitis.

Varieties and Etiology.—Chronic pharyngitis may assume one of four types, viz.:

1. *Diffuse inflammation* of the mucosa. This type sometimes follows the acute form.

2. Hypertrophy of the lymphoid tissue surrounding the pharyngeal follicles, or *follicular pharyngitis*. As the lymphatic structures are more numerous on the lateral walls of the pharynx the nodular thickenings are most prominent in these locations. The condition is sometimes referred to as *pharyngitis lateralis hypertrophica*. Mouth-breathing, nasal or tonsilar infections, are factors in the production of this type of chronic pharyngitis.

3. *Exudative pharyngitis* is a modification of the follicular or granular type. The secretion instead of being clear becomes opaque and of a pasty consistence, as in follicular tonsillitis.

4. The so-called *pharyngitis sicca* is not a true inflammation but a dry, glazed condition of the mucous membrane due to diseased conditions of the nose or tonsils, as atrophic rhinitis, sinusitis, nasal obstruction, chronic tonsillitis, etc. The various anemias predispose to it. Perhaps a more appropriate name for the condition would be pharyngea sicca.

Symptoms.—The patient usually complains of a sense of fulness in the throat, with slight pain or discomfort, and is troubled with hawking and expectorating on rising in the morning. When the second stage (simple follicular or granular) is reached, the voice tires more easily, and the throat becomes more painful after slight use. The voice after a time becomes husky, necessitating frequent "hemming" to keep it clear. Cough is a troublesome symptom in all varieties, but more especially in the follicular and exudative types. The hearing is sometimes impaired by plugs of mucus in the Eustachian tubes, and more rarely by extension of the inflammation into the middle ear.

Treatment.—It is well to *begin* the treatment by the administration of a saline cathartic, followed with iron, tonic doses of quinin, nux vomica, phosphorus, etc. The use of spirits, tobacco, and an over-rich diet should be interdicted. If the *uvula* is elongated, it should be amputated at about its middle portion. The local use of *vegetable astringents*, glycerite of tannin, etc., is of value in the simpler cases.

In the *follicular variety* the mineral astringents, as the perchlorid of iron, nitrate of silver, and the tincture of iodin are of greater value than the vegetable astringents. The truth is that nothing short of caustic applications to the enlarged lymphoid nodules is of lasting benefit. London paste, lunar caustic, chromic acid, etc., have given way to the more convenient and thorough *electric cautery*. The small platinum-wire point can be limited to a much smaller area than the mineral caustics. Before using either the chemical or electric cautery the pharynx should be cocainized.

In the *exudative type* the opaque secretion, with a small sharp spoon, should be squeezed or scraped out of the follicles which should then be lightly cauterized with the fine platinum electrode brought to a red heat.

Pharyngea sicca, caused by general anemia, should suggest the administration of appropriate remedies for the anemia. If it is due to stenosis of the nose, nasal surgery should be resorted to for its relief. Local applications of a mildly aromatic and stimulating nature are grateful to the patient. They are best applied with an atomizer. Gargles are of little or no value unless it is in the diffuse variety, which may be regarded as subacute in character.

Chronic Nasopharyngitis.—**Synonyms.**—Chronic epipharyngitis; nasopharyngeal catarrh.

Etiology.—This is a chronic inflammatory condition of the vault of the pharynx which is usually associated with, and is probably secondary to, some form of intranasal disease. It is also associated with certain other morbid states of the nasopharynx.

Inflammatory conditions of the nasopharynx is rarely a primary affection. It is almost always secondary to intranasal affections and adenoid disease of the nasopharynx. The following are the *diseases* usually responsible for the condition:

1. *Nasal stenosis* from hypertrophy of the turbinated bodies, more especially of their posterior extremities.
2. *Polypi* causing stenosis and rhinitis.
3. *Empyema* of the *posterior ethmoidal cells* discharging posteriorly.
4. *Empyema* of the *sphenoidal sinuses* discharging posteriorly.
5. *Dry rhinitis.*
6. *Atrophic rhinitis.*
7. *Simple chronic rhinitis* with collapse of the erectile tissue as found in anemic persons.
8. *Pus discharged through the Eustachian tubes* during otitis media suppurativa.
9. *Disease of the pharyngeal bursa.*

Symptoms.—They will depend upon which of the foregoing causes is operative in its production. In addition to the nasal, ear, and other symptoms, the patient complains of a discharge (mucus and mucopus) from the upper pharynx. This is usually described as "a dropping." If the pus is from the nose, it usually has a thick yellow quality; while if it is from the pharyngeal bursa, it is more fluid. It may accumulate and dry during the night, causing the formation of crusts.

Diagnosis.—The diagnosis should be addressed more to differentiation of the causes than to the local condition *per se*. The local condition is to be regarded as a symptom of one or more of many causes,

Prognosis.—The prognosis depends upon the ability of the examining physician to properly locate the cause and relieve the same. If the intranasal or other causes are removed, the cure of the postnasal irritation is reasonably certain.

Treatment.—In view of the foregoing statements it is apparent that local washes, sprays, and astringent applications afford but temporary relief in the great majority of cases. *The treatment must be addressed to the removal of the intranasal and other causes.* The treatment of the nasal accessory sinus, ear, and adenoid diseases are considered elsewhere, and will not be further elaborated here.

Local alkaline and antiseptic washes used with the postnasal syringe afford marked relief. Seiler's or Dobel's solutions are easily obtained and are well suited for this purpose. Astringent applications to the nasopharynx of glycerotannin, argentic nitrate gr. x to xxx to the ounce are regarded by some as efficient remedies. The following mixture, sometimes spoken of as the Vienna mixture, has proven a valuable addition to the local treatment in the practice of the author:

℞—Iodin. cryst.	gr. x
Potass. iodid.	gr. xxx
Glycerin.	℥j—M.

Sig.—To be applied to the nasopharynx with a cotton-wound applicator after cleansing with an alkaline antiseptic solution.

If alcohol is used to excess, it should be interdicted. The clothing should receive proper attention, and morning plunge baths recommended for their tonic effect.

Acute Tonsillitis.—**General Description.**—Under this caption will be considered what is sometimes described as several distinct diseases, *i. e.*, (*a*) *acute superficial tonsillitis;* (*b*) *follicular tonsillitis;* and (*c*) *parenchymatous tonsillitis.*

These conditions are much the same except in degree, the nature of the exudate, and the point of greatest inflammation. Clinically, the various conditions are often associated at some stage of the process and the symptom-complex can be best studied as an entity.

When the crypts, of which there are ten to fifteen in each tonsil, are chiefly involved, they become distended with epithelial cells, leukocytes and microörganisms. Streptococci and staphylococci are the microörganisms chiefly associated with this type of tonsillar inflammation.

Etiology.—The parenchymatous inflammation may be attended by hypertrophy of the tonsillar tissue, especially after repeated attacks. In children slight irritations may cause hypertrophy of the lymphoid tissues. Such irritations may arise during a cold, scarlet fever, measles, influenza, diphtheria, and other exanthematous fevers. It should be remembered, however, that all children who are thus exposed do not have enlargement of the tonsils.

Tonsillitis is sometimes epidemic over considerable areas. Exposure to wet and cold, the sudden changes of temperature, insufficient clothing and protection to the feet, are sufficient to predispose to an attack of acute tonsillitis in many individuals.

The disease is most common at the period of adolescence and young adulthood, although it may occur at any age.

Symptoms.—These vary with the degree, type, and location of the focal points of inflammation. In the *simple diffused form* there is slight swelling and redness accompanied by a feeling of discomfort upon deglutition. The pain is often described as a prickling one, as from the presence of a fish bone or other small sliver in the fauces.

In the follicular or lacunar type of tonsillitis one tonsil usually becomes involved before the other. The crypts are filled with a purulent exudate composed of epithelium, leukocytes, and bacteria. The masses of exudate are yellow in color and may project from the lacunar openings. The pain may not be so severe as in the so-called diffuse stage.

If the crypts and surface of the tonsil are lined with a fibrinous exudate, the symptoms are apt to be much more severe. The parenchyma of the tonsil, together with the contiguous structures—*i. e.*, the pillars and uvula—are often inflamed and edematous. This *fibrinous type* was formerly often mistaken for true diphtheria. The pseudomembrane is not, however, adherent to the surface of the tonsil as it is in diphtheria. A bacteriologic examination of the tonsillar secretion will aid in making a correct diagnosis.

The temperature may rise from 101° to 105° F.

General Treatment.—Rest in bed, fluid or semisolid diet and free catharsis are the essentials of the general treatment. Salicylates, aspirin, quinin, tincture of aconite, perchlorid of iron, etc., have been recommended.

Local Treatment.—A 25 to 50 per cent. aqueous solution of nitrate of silver is a most excellent *local application* in the acute stage of the superficial inflammation. The application of the nitrate of silver in this strength is attended by some danger, as a drop of it flowing down upon the larynx would excite most violent laryngeal spasms. Extreme care should be taken to remove the excess of fluid from the cotton mop before applying it to the crypts and the surface of the tonsil. In the author's experience only one or two applications have been required to abort the inflammation if seen in the early stage.

Other local remedies of value in the later stages are 25 per cent. argyrol, tr. benzoin co., mentholated oil, etc. Hot alkaline antiseptic gargles may be used.

Peritonsillar Abscess; Quinsy.—Pathology.—Peritonsillar abscess or abscess of the tissue surrounding the tonsil is usually unilateral. There is a preceding tonsillitis in most cases. The soft palate, uvula, and pillars become congested and often edematous. If permitted to progress, spontaneous rupture usually takes place through the anterior pillar or velum, through the supratonsillar fossa, or through the posterior pillar.

Symptoms.—If the inflammation is chiefly centered in the loose connective tissue surrounding the tonsil, the movements of the lower jaw are very much interfered with. The soft palate and uvula are usually edematous and nasal respiration may be impaired. The secretions are profuse and stringy and may be composed of mucous or mucopurulent matter. The patient has a stupid, anxious cast of countenance, the mouth being open and the saliva driveling therefrom.

Severe pain which steadily grows worse in the region of the tonsil and dysphagia are prominent symptoms.

Upon *inspection*, one side of the fauces will be found to be very much swollen and edematous, and covered with heavy mucus which is mixed with epithelial cells, cocci, and leukocytes.

Treatment.—Bosworth recommends that the patient be provided with a saucer of the bicarbonate of soda into which he should dip his moistened finger at frequent intervals and plaster it on the inflamed tonsil. He claims that it reduces the amount of pain and exerts a favorable action on the course of the disease. If marked depression is present, the tincture of iron and alcoholic stimulants should be given.

When the disease has progressed to abscess formation, the point of greatest fluctuation should be located and freely incised with a bistoury. It usually points above and anterior to the tonsil. A small pointed bistoury should be used, making a free incision parallel with the free border of the anterior pillar, so as to avoid cutting the fibers of the palatine muscles. The edges of the wound should be separated with a pair of forceps to facilitate the escape of pus. It is not best to wait for spontaneous rupture of the abscess, as it may burrow beneath the deep fasciæ of the neck and lead to serious complications.

If the incision through the anterior pillar fails to reach the abscess, the surer method of reaching the abscess by dissecting the anterior pillar from the tonsil may be employed.

Technic.—(1) Inject a 2 to 5 per cent. solution of cocain or novocain through the anterior pillar into the peritonsillar tissue. (2) With a tonsil forceps pull the tonsil medianward and forward and make an incision at the junction of the anterior pillar and the tonsil. (3) Introduce a blunt dissector or a blunt-tipped dressing forceps through the incision and separate the tonsil from the peritonsillar tissue until the abscess cavity is reached.

Vincent's Angina.—**Etiology.**—The predisposing factors are lowered bodily resistance, diseased tonsils, teeth, and gums. It frequently occurs after measles, scarlet fever, diphtheria, whooping-cough, etc. The exciting cause is the fusiform bacillus and spirillum of Vincent. The fusiform rod bacillus is boomerang-shaped, with tapering ends. It is slightly motile. The spirocheta is slender and undulating. Both organisms are Gram-negative.

Pathology.—The gray or grayish-white membrane of Vincent's angina is a pseudomembrane, due to necrosis of the superficial layers of the epithelium.

If the membrane is removed, an ulcerated area is seen which bleeds easily. The spirochetæ are confined to the surface of the tonsil or its crypts (Wingrave). The point of attack is usually the tonsil, but the infection may spread to the pharynx, uvula and soft palate.

Symptoms.—Two forms of the disease are seen. In the first or mild type the infection is confined to the tonsils. The patient complains of chilly sensation, general malaise, pain in the tonsillar region on swallowing, and slight fever, rarely above 100° F. This form runs its course in a few days.

In the second or severe form the extratonsillar structures are usually involved. Extreme prostration may occur. The pain is severe. The temperature, usually low, may reach 105° F. The breath is very fetid and is a very prominent symptom. This form may last for weeks. Some even result fatally. The diagnosis is made from finding the organisms. Both smear and culture should be made.

Treatment.—In the treatment of Vincent's angina various antiseptics have been used, such as tincture of iodin, silver nitrate, trichloracetic acid, chromic acid, formalin, etc. Salvarsan has been used both locally and intravenously. Citron uses a trituration of salvarsan in glycerin (0.1 to 5 cm.) applied locally.

Chronic Tonsillitis.—**Etiology.**—The hypertrophic form of chronic tonsillitis is essentially a disease peculiar to childhood, rarely having its beginning in adult life. With the passing of the period of adolescence the tendency to hypertrophy disappears. Sclerosis and shrinkage of the tonsillar substance begins and may continue until it is quite firm and gristly. The chronic lacunar type of tonsillitis occurs more frequently in adults.

Either type may be due to repeated attacks of acute tonsillitis. It may follow the acute exanthemata and other acute infections, influenza, etc. There is undoubtedly a hereditary tendency in many cases.

Pathology.—Three general classifications may be made, viz.: (1) *True hypertrophy*, in which all the elements composing the tonsil are increased in size and number. The lymphoid nodules are particularly thus affected. (2) *Hyperplasia*, or sclerosis, in which the stroma or connective-tissue framework of the tonsil is increased. As the process advances the bloodvessels and lymphoid nodules are diminished in size. The tonsil, instead of being soft, as in the hypertrophic variety, is firm and unyielding to the touch. (3) The *lacunar type* is characterized by a ragged appearance, and has been called the "honey-combed" tonsil. There are normally from ten to fifteen crypts in each tonsil. They become enlarged from being distended by accumulations of epithelial cells, leukocytes, and débris. A probe can be passed into them for a half-inch or more. In the lacunar or "ragged" tonsil they are sometimes found to communicate with each other. This may be due to the absorption of the intervening wall by pressure necrosis; or to a caseous degeneration of the lymphoid nodules, leaving cystic cavities where true parenchyma once existed.

Sometimes the pillars of the fauces are adherent to the tonsils and should be dissected loose before attempting the removal of the latter.

Symptoms.—In the hypertrophic form the symptoms are mainly those due to mechanical interference with the normal respiration.

The muscles of deglutition are hampered in their action. Hyperemia of the larynx is induced, and the voice may be husky, toneless, and easily fatigued. The tensor palati and levator palati muscles are obstructed in their action, and as they largely control the patency of the Eustachian tubes, the proper aëration of the tympanum is not accomplished.

Hypertrophy of the pharyngeal, lingual, and nasal lymphoid masses or tonsils is commonly associated with enlargement of the faucial tonsils.

As a *result* of the obstruction to nasal respiration the patient snores during sleep. The facial expression is somewhat dull, and the mind is often as dull as the face betrays. Aprosexia, or difficult attention, is a common symptom. The chest is often malformed, pigeon-breast being the usual type. The cervical glands are frequently enlarged.

The sense of smell and taste are sometimes impaired.

In the lacunar type of tonsillitis the patient frequently complains of a bad taste, especially in the morning, or of an offensive breath. Acute exacerbations are common, although many patients will give a history of no attacks of acute tonsillitis or sore throat.

Objectively the tonsils appear as roundish masses projecting from between the anterior and posterior pillars of the fauces. In the hypertrophic type they are of a pinkish-red color, while in the hyperplastic type they are much paler. In the lacunar variety they appear as ragged masses or, if seen in the earlier stage, as swollen tonsils, with the crypts filled with an ivory-colored concretion.

Treatment.– Numerous *local remedies* applied to the tonsils for the purpose of causing them to shrink have been tried from time to time. They, however, do no more than reduce the hyperemia of the mucosa, the hypertrophic process being in nowise arrested.

The tincture of iodin has been applied to the tonsils and to the skin over the angle of the inferior maxilla with the vain hope of arresting the tonsillar growth.

Ignipuncture is mentioned only to be condemned, as it closes up the crypts and retains the secretions. It is performed as follows: (1) The tonsil is painted at intervals of one minute with a 10 per cent. solution of cocain until local anesthesia is produced. (2) The electrode (Fig. 137), a simple pointed one, should be placed against the tonsil while cold, and the current connected, so regulated previously as to bring the platinum point to a red heat. The electrode while thus heated should be plunged to the depth of half an inch into the tonsil. This procedure should be repeated three or four times at the same sitting.

FIG. 137.—Galvano-cautery electrode.

Where operation is refused, the *lacunar variety* of enlarged tonsils may be treated as follows:

Remove the accumulations of epithelial cells and leukocytes from the crypts by squeezing the tonsils; or by means of a tonsil curette or swab, then paint the crypts with a 25 per cent. silver nitrate solution or a 20 per cent. trichloracetic acid solution.

All the foregoing *local* methods of treatment are but makeshifts, to be used only when the following radical and more reliable method is refused.

Tonsillectomy.—Various methods have been devised for the complete removal of the tonsil and its capsule. Among them may be mentioned the dissection method with a straight or curved knife, the dissection and snare, the scissors dissection, the guillotine methods by means of Sluder's guillotine or one of its modifications.

Tonsillectomy by Means of the Ballenger-Sluder Tonsillectome.—Two forms of the instrument are made, one with a semisharp blade (Fig. 138) and the other with two blades, one sharp and the other dull. Both instruments have the scissors handle, which greatly facilitates the operation and requires very much less muscular exertion to sever the tissues.

The **technic** as employed by the writer is similar to that employed by Sluder, although differing in some details. When the first-mentioned instrument is used, the technic is as follows:

In children a general anesthetic is almost invariably given, as the fright and mental shock of an operation under local anesthesia sometimes results in an injury to the nervous system. A local anesthetic is employed for adults.

When local anesthesia is used, a preliminary swabbing of a 5 to 10 per cent. cocain solution to the anterior pillar may be made, although not essential. Three or four injections of 5 to 10 minims each of a 0.5 to 1 per cent. cocain solution with 8 drops of adrenalin chlorid (1 to 1000) to the ounce, added, may be injected into the tissue surrounding the tonsil.

In removing the right tonsil the guillotine is held in the right hand and inserted through the left side of the patient's mouth until the distal portion of the instrument is in front of the posterior pillar and behind and slightly below the tonsil (Fig. 139).

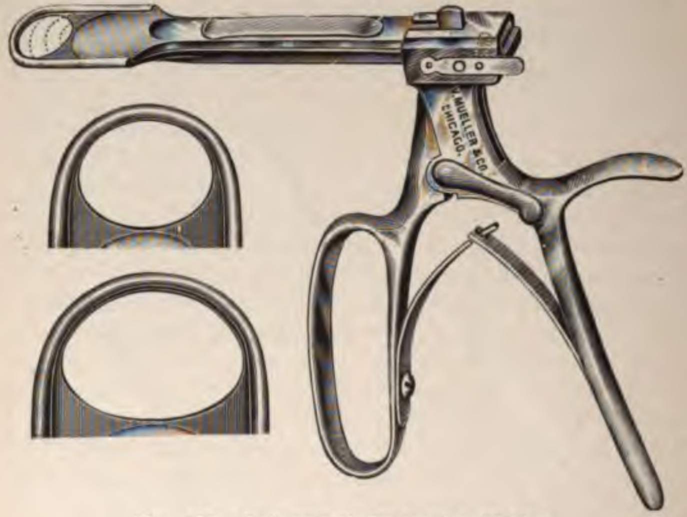

FIG. 138.—Ballenger-Sluder tonsil guillotine.

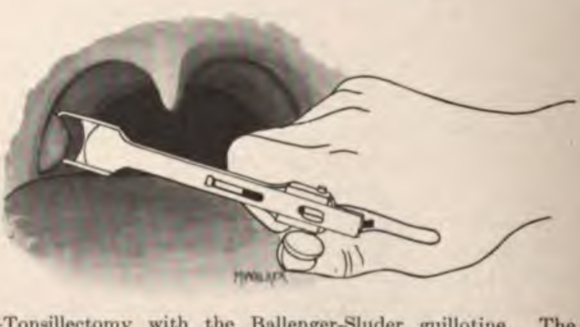

FIG. 139.—Tonsillectomy with the Ballenger-Sluder guillotine. The tip of the instrument is placed behind the tonsil and in front of the posterior pillar.

The tonsil is now pushed upward and forward until there is a distinct bulging over the anterior pillar. In children where the tonsil is a so-called protruding one and is not buried, no displacement is necessary, as the tonsil will readily become engaged in the ring of the instrument without force or pressure. In an adherent, buried tonsil more force will be required to displace it.

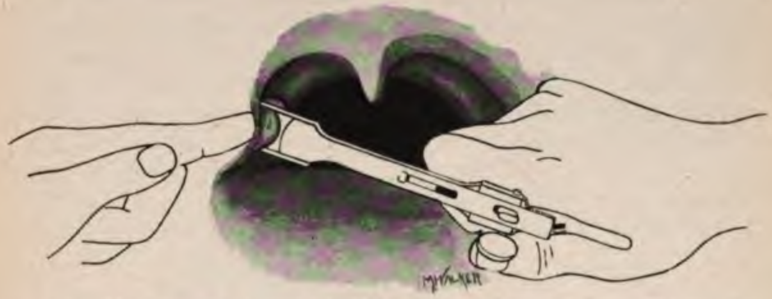

Fig. 140.—Tonsillectomy with the Ballenger-Sluder guillotine. The tonsil is drawn forward and upward. Counterpressure is furnished by the index finger of the unengaged hand.

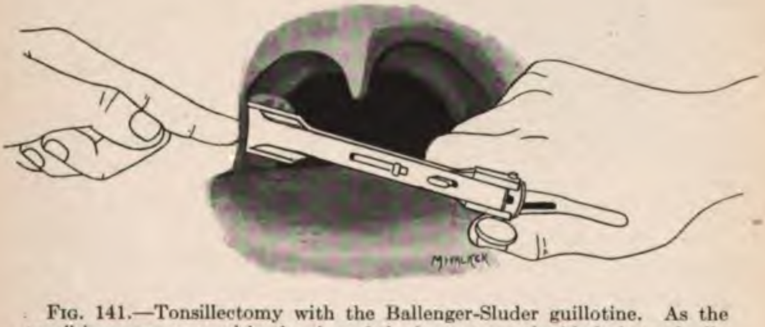

Fig. 141.—Tonsillectomy with the Ballenger-Sluder guillotine. As the tonsil becomes engaged in the ring of the instrument the blade is gradually pushed home.

Having displaced the tonsil forward and slightly upward and maintaining it in the new position with firm pressure, the index finger of the left hand is inserted and pressure is made over the bulging anterior pillar until the tonsil is felt to pass through the ring of the instrument (Fig. 140). As the tonsil is engaged and as the pressure is made on the anterior pillar the handle of the instrument should be brought over to the right side of the patient's mouth (Fig. 142). The blade of the instrument is gradually pushed down as the tonsil passes through the ring of the instrument. The pushing of the tonsil through the ring, the bringing of the handle of the instrument to the right side of the patient's mouth and the driving of the blade home are all done at the same time. Care should be observed to have the blade of the instrument sever the mucous membrane at its attachment to the tonsil and not away from the tonsil as may easily be done when an adherent and buried tonsil is present. By this method it is almost impossible to injure the posterior pillar unless it is fused with the tonsil.

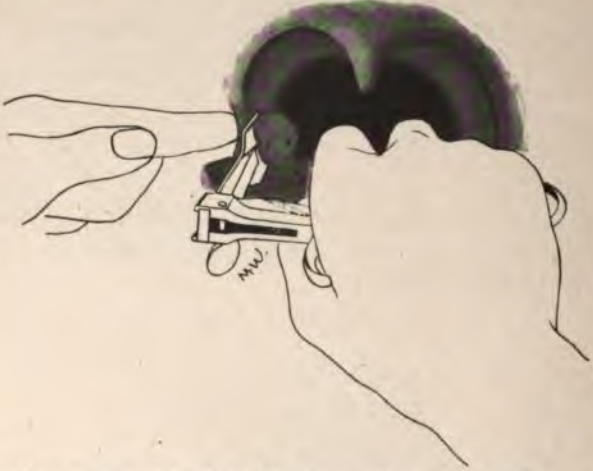

Fig. 142.—Tonsillectomy with the Ballenger-Sluder guillotine. As the blade is pushed home, the handle of the instrument is brought over to the side of the mouth from which the tonsil is being removed.

The left tonsil is removed as described for the right except the instrument should be held in the left hand instead of the right and should be introduced from the right angle of the patient's mouth instead of the left.

The objections made to the guillotine type of instrument are that the anterior pillar is sometimes cut and that the plica triangularis is removed. The first objection is not based on experience, as the blade of the instrument may be made to sever the attachment of the pillar as close to the tonsil as desired, indeed the tendency of the instrument is to hug the capsule of the tonsil. Concerning the second objection, namely, the removal of the plica triangularis. This is rarely done, as displacing the tonsil forward and upward puts the plica on a stretch and hence is not removed.

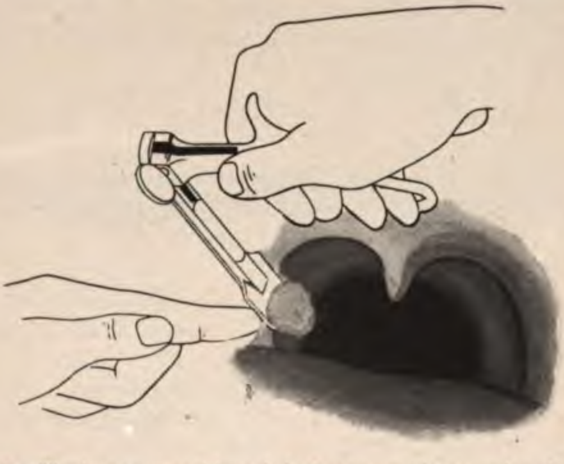

FIG. 143.—The incorrect position of the instrument in removing the tonsil.

The double-bladed instrument (Fig. 143) is used as is the single-bladed one, except before severing the tonsil the dull blade is pushed home with the ratchet handle and left in position for from five to ten minutes or until clotting has occurred, when the tonsil is severed by screwing down the sharp blade.

Instrumental Dissection of Tonsil.—Many methods, differing somewhat in detail, have been devised for the dissection of the tonsils. The following method is used by Fletcher and Sher:

Technic.—In order to have the operation as bloodless and as painless as possible the tonsils are injected with the following formula: cocain, gr. 1; adrenalin, minim 20; normal salt solution, minim 80. About 10 minims of this solution is injected into each tonsil with a hypodermic syringe, the needle of which is bent at an angle of about 65 degrees. Blisters are made directly beneath the membrane. The first blister is over the insertion of the anterior pillar, the second at its centre, the third just above the orifice of the superior crypt and the fourth at the upper half of the posterior pillar. The solution is used in both local and general anesthetics. In the local anesthetic about 5 minims of a 1 per cent. novocain solution is injected behind the tonsil.

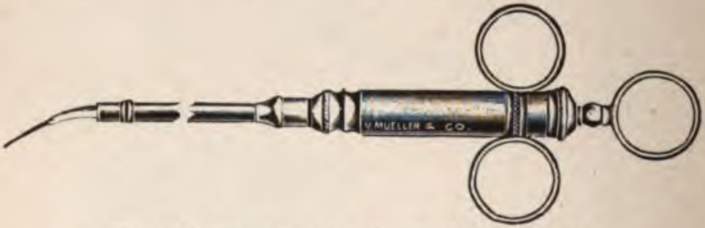

Fig. 144.—Street's syringe for injecting the tonsil.

Under a general anesthetic the patient should lie flat upon an ordinary operating table, the mouth well opened by use of a mouth gag. First one and then the other tonsil is injected as described. The tonsil is seized and lightly drawn out. Just enough traction should be made to unfold the membrane which passes over the face and posterior border of the anterior pillar. This makes a distinct tuck which extends upward and backward following the arch of the palatoglossus muscle. There is a distinct demarcation between this membrane and the tissue through which the crypts open. This is the line to follow in dissecting the membrane from the tonsils.

The incision is started at the lowest point of the plica triangularis and carried upward in a curved line following the outline of the tonsil to a point just over the mouth of the superior crypt.

The incision is then carried backward and downward, separating the tonsil from the posterior pillar, to the lowest part of the tonsil. The second incision is begun where the first was started and carried downward to meet the end of the first. This completes the cut. The membrane is freed from the tonsil by pushing the membrane over the upper pole of the tonsil with the blunt end of a knife until the bluish aponeurosis of the superior constrictor is seen. The dissection is completed by using an instrument shaped like a Sims uterine scissors, sharp on both sides. The dissector is introduced where the bluish aponeurosis was observed and with a slight downward and backward push the upper part of the tonsil is freed. The dissection is continued until the tonsil falls out of the pyriform fossa. The removal of the tonsil is then completed by means of a snare.

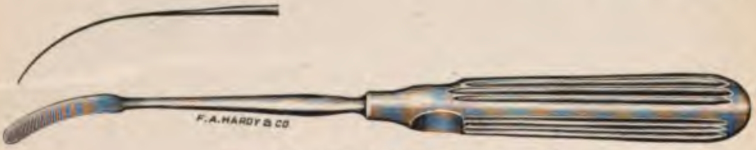

FIG. 145.—Sher's tonsil dissector.

Stein's Tonsil Snare.—The method of using the Stein snare (Fig. 146) is similar to that of the Sluder blade, namely, grasping the instrument in the right hand for operating on the right tonsil when working in front of the patient, as is usually done. The fenestrated end is engaged behind and below the lowest part of the gland which is then drawn forward and outward in a hook-like motion until the gland appears caught behind the anterior pillar. The index finger of the unengaged hand can then be placed against the pillar to act as a fulcrum against which the gland is held as the instrument is rotated toward the side being operated, thus engaging the anterior and superior portions of the tonsil with the fenestrum. The wire is now quickly tightened by working the wheel and ratchet until it is engaged behind the gland and its capsule when it is tightened more slowly. If sufficient time is taken, say three or four minutes, Stein claims no bleeding whatsoever follows. Stein claims the following advantages for his instrument: strength and durability; adaptability to any hand; adequate lifting power; easy control and operation with no obstruction of view.

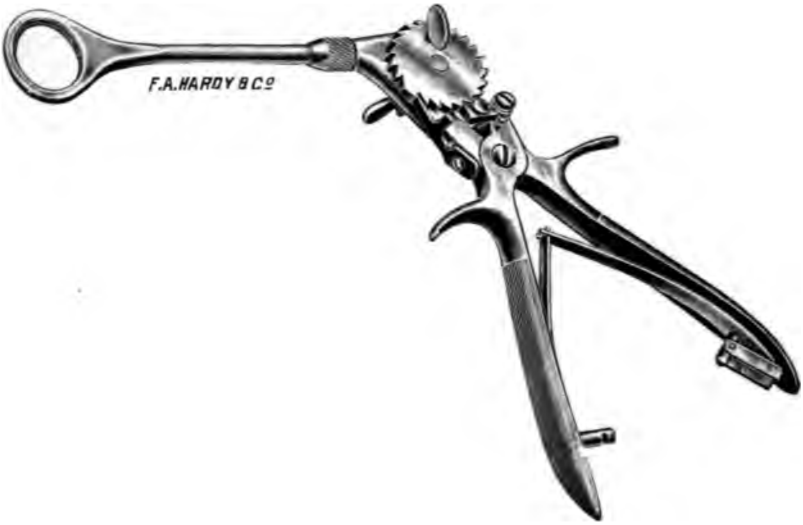

FIG. 146.—Stein's tonsil snare.

Foreign Bodies in the Tonsils.—The tonsils when enlarged present a surface with numerous pits or lacunæ. They lie between the anterior and posterior pillars of the fauces, hence they present favorable conditions for the lodgment of foreign bodies, such as fish bones, sharp splinters of bone or wood, pins, needles, and other sharp substances.

The patient should be directed to relax his throat as much as possible during the examination, as otherwise the tense pillars of the fauces may conceal the foreign body from view.

Mycosis Leptothricia (*Hyperkeratosis of the Tonsil*).—**Etiology.** This is a disease, or rather a form of fungous growth, attending certain morbid states of the fauces, but more particularly the tonsils. The growth or fungus belongs to the schizomycetes group. A number of kinds of leptothrix are found in the mouth and fauces. They are regarded as non-pathogenic. They are slender rods staining with iodin solutions. They pile up on end, forming tufts of the prickly wart style. When seen in the acute stage they are of a soft creamy consistency, and may be wiped from the membrane easily. When they are of long standing, they are firmly attached to the epithelial layer of the mucosa and are difficult of removal. Lacunar disease of the tonsils strongly favors their development. Live tissue has no attractions for them, while dead epithelial cells are their favorite tissue attachments. Keratosis of the tonsils is therefore a strong predisposing factor in their

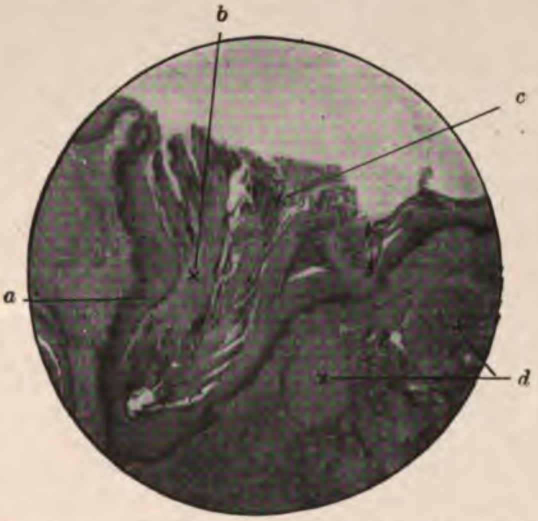

FIG. 147.—Hyperkeratosis, showing the typical appearance under low power. The horny mass is growing from a comparatively small area of the cryptal epithelium, and the plug shows the ordinary fraying of its edges: *a*, cryptal epithelium; *b*, horny material; *c*, masses of bacteria; *d*, follicles. (Wood.)

development. The author has often seen mycosis buccalis at the point where the upper and lower teeth come together, in other words, where keratosis occurs as a result of friction.

The size of the tufts varies from that of a pin-point to that of a small wart.

Treatment.—The disease is usually self-limited, from six months to three years being required for it to run its course. Mouth washes and gargles of chlorat of potassium and other antiseptic solutions exert but slight influence on the growth. Equal parts of guaiacol in olive oil or sweet oil of almonds applied locally seems to be of decided benefit. Salicylic acid has its enthusiastic supporters, but the facts are that nothing short of the removal and cauterization of the points of attachment is of positive value in their complete eradication. When they are once thoroughly removed, their return is still quite probable.

If the tonsils are enlarged, their removal exerts a favorable influence on the growth.

Adenoids.—**Synonyms.**—Hypertrophy of the Pharyngeal Tonsil; Postnasal Vegetations. These and other names have been used to designate enlargement of the normal lymphoid masses on the vault and posterior superior wall of the nasopharynx (Fig. 148).

Etiology.—*Age* is of prime importance in the causation of hypertrophy of the pharyngeal tonsil. Most cases develop before the age of puberty, at which time they usually begin to atrophy.

Cases are reported in persons more than seventy years old. They are rare, however, in advanced years, and when present may be of rather recent growth from inflammatory causes.

In most persons affected in youth only a trace is left after the thirtieth year. Just prior to puberty a retrograde metamorphosis in the form of colloid degeneration usually affects the pharyngeal tonsil.

Sex does not appear to be a decisive factor in the production of this disease.

Either *constitutional conditions* are a cause of postnasal adenoids or the adenoids produce the constitutional conditions. They are usually associated; writers differing as to which is cause and which is effect. Most authorities, however, agree that constitutional conditions are potent predisposing causes.

Heredity has great influence in the development of adenoids, as the growths often appear in several members of the same family.

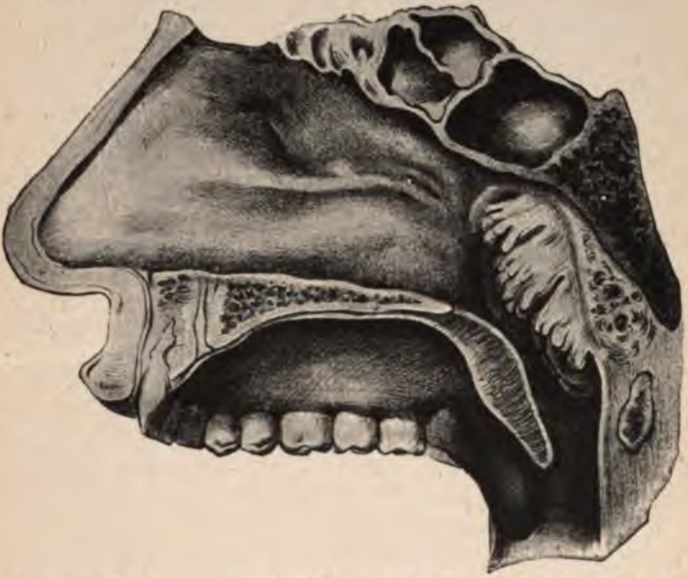

FIG. 148.—Anteroposterior section of the head of an adult, showing the situation and gross structure of hypertrophy of the lymphoid tissue of the nasopharynx. (Zuckerkandl.)

Climate perhaps in a measure accounts somewhat for the above distribution. It is certain that humid, cold climates favor adenoid growths in children, while warm climates minimize the hypertrophic tendency.

Inflammatory action in the nose and nasopharynx, as seen during an attack of the exanthema (specific fevers), play an important *role*. Common colds are perhaps the most responsible for exciting lymphoid hypertrophy in the nasopharynx.

Nasal obstruction increases the tendency to hyperplasia of the lymphoid tissue in the nasopharynx. A high-arched palate is frequently associated with a large adenoid and is frequently responsible for the apparent lack of improvement in the nasal breathing following the thorough removal of the adenoid mass.

Histopathology.—There are normally groups of lymphoid tissue or glands in the vault and posterior wall of the nasopharynx. They extend from the *bursa pharyngea* in the median line across the upper and back part of the pharynx. The tissue is of a low embryonic type, and is excited to hypertrophy under slight irritation during childhood. The nodules are of irregular shape and distribution. The crypts of the pharyngeal, unlike those of the faucial and lingual, tonsils are absent or ill developed. The surface is covered by a single layer of ciliated columnar epithelium, except where contact and movement against opposing structures have caused their disappearance. Mingled with the ciliated epithelium are numerous chalice cells, which in a measure account for the abundant thick mucous secretion in the nasopharynx. There are no true mucous glands, but there is a single row of lymph nodules near the surface. In the deeper layers there is a heavy reticulum, the meshes of which are filled with small lymphoid cells each containing one or two nuclei.

The gross **morbid appearance** of the growth as seen *in situ* is usually a cushion-like mass, slightly nodular. As has been aptly expressed by Meyer, when the finger is introduced into the nasopharynx this mass gives a sensation like "pushing the finger into a bunch of earthworms." More rarely the growth is very nodular, or even fimbriated, as is shown in most drawings in text-books.

Symptoms.—The *physiognomy* affords the most striking and at the same time characteristic symptom of the disease when advanced. The mouth is open, the nasolabial furrow absent or slightly developed, the bridge of the nose widened, the cheeks flattened, the inner canthi drawn down, the root of the nose puffy and edematous, and the eyelids slightly drooping. Taken *ensemble*, the face has a stupid expression which cannot but attract attention. The alæ nasi are depressed at the angle between the superior and inferior lateral cartilages.

The *objective signs* in addition to those given under physiognomy are those to be seen or felt in the pharynx, nasopharynx, fauces, nose, and chest. The pharynx usually presents a granular or nodular surface covered with frothy mucus. The lymph nodules are those involved in follicular pharyngitis elsewhere described. They are somewhat edematous and succulent in appearance. Occasionally there is pharyngitis sicca, which is not attended by rhinitis sicca. The uvula is sometimes edematous. The pillars and velum are congested and of a dusky red color. Pigeon-breast is an associated condition which has been variously explained.

Aprosexia, or *mental dulness*, is in most cases more or less present. Various theories have been advanced in explanation of it.

Sleep is disturbed, the child tosses in bed, throwing off the covers, and usually snores on account of the mouth-breathing; each inspiration causes the relaxed membrane to vibrate. The mental state during sleep is disturbed by dreams and nightmare. Upon awakening, these mental impressions often persist for some minutes, giving rise to what has been described as slight delirium. The snoring sometimes terminates in laryngeal stridor, and even in mild general convulsions. Upon awakening, the mouth and throat are dry. General malaise is often experienced in the morning, but disappears as the day advances.

Reflex phenomena often attend, if they do not result from, the adenoid growths. Hay fever, paroxysmal sneezing, asthma, chorea, epilepsy, torticollis, headache, enuresis, genital irritation, exaggerated peristalsis of the intestines resulting in prolapse of the rectum have been reported as concomitant symptoms of the presence of postnasal adenoids.

Hearing.—Disturbances of the function of hearing is one of the most common attendant symptoms of adenoids. Fully 60 per cent. of all persons with hypertrophic adenoids in the pharynx have more or less interference with audition.

Diagnosis.—In addition to the above symptoms the diagnosis may be verified by one or more of the following procedures:

1. *Anterior Rhinoscopy.*—Occasionally it is possible to observe the adenoid mass by anterior rhinoscopy.

2. *Posterior Rhinoscopy.*—In most instances a satisfactory view of the epipharynx can be obtained by posterior rhinoscopy providing the confidence of the child is obtained. This is the method that should be used if possible, as the pain and fright from a digital examination destroys all confidence that the child may have in the examiner.

3. *Digital Examination.*—The examiner stands on the patient's right side and holds the patient's head with the left arm. To prevent the child biting the examiner's finger it is necessary to hold the cheek between the open jaws. While the mouth is opened widely the index finger (previously lubricated with vaselin) of the right hand is passed quickly against the posterior wall and then gently pushed upward into the epipharynx where the soft adenoid mass is felt if present.

Treatment.—The *medical treatment* is of but little value except as it affords temporary relief of some of the distressing symptoms. Alkaline and aromatic washes and sprays to the nose and nasopharynx remove accumulated secretions and diminish the congestion.

Alkaline washes may be pursued to relieve certain distressing conditions while waiting for the operative treatment.

Surgical measures are the only ones that are of permanent value. Various procedures, as scarification, superficial scraping, etc., to excite bleeding have been tried with the hope of affording permanent relief. Inasmuch as the condition is one of hypertrophy and not of congestion, such measures are inadequate. The *removal of the growth* is essential to complete success.

The **chief indications** according to Washham for the removal of the adenoids are: (1) well-marked signs of nasal obstruction, such as mouth-breathing, adenoid expression of countenance, alteration of voice; (2) deafness, or oft-recurring attacks of deafness, the result of middle-ear disease; (3) continually recurring head-colds, or nasal, laryngeal, or bronchial catarrh; (4) paroxysmal attacks of sneezing, hay fever, laryngismus, or spasmodic asthma; (5) defective physical and mental development, chronic tuberculous enlargement of the cervical glands.

Various instruments have been devised for the performance of this operation, among which are: Meyer's ring knife, to be used through the nose; Loewenberg's or Brandegee's forceps, curved so as to be used through the mouth and introduced into the nasopharynx; the Gottstein or Barnhill curette (Fig. 150), to be introduced into the nasopharynx through the mouth; the La Force adenotome, etc.

The **anesthetic** may be local or general. Cocain and novocain solutions may be applied *locally* through the nostrils or through the mouth by means of curved cotton carriers saturated with the solution. Anesthesia more or less complete may be thus produced. According to my own experience, it is not a very satisfactory method.

General anesthesia is preferable and may be induced by the use of nitrous oxide or ether. Chloroform seems to act unfavorably in children with adenoids and is to be condemned.

The *instruments* used by the writer are the Whitehead mouth gag (Fig. 151), the La Force adenotome, the Barnhill curette, tongue depresser, and curved sponge holders.

The Position of the Patient.—In adults under local anesthesia the upright position may be used. When a general anesthetic is given, a dorsal position is safer and more satisfactory, although many operators prefer to have an assistant hold the anesthetized patient in the upright position. If the latter method is used, an assistant takes the child on his lap and crosses his legs over those of the child. The folded arms are held with his left hand and arm, while the head of the child is held against the right shoulder of the assistant with his right hand grasping the forehead.

The *technic* as employed by the writer is as follows: If a general anesthetic (dorsal position) is used, a mouth gag (Whitehead) is inserted. With a local anesthetic no mouth gag is required. The tongue is held down with a tongue depressor and a La Force or Collum adenotome (Fig. 149) of suitable size is introduced into the fauces. To introduce the adenotome behind the soft palate it is sometimes necessary to turn the tip outward so that one corner of the instrument will first engage behind the soft palate. The adenotome should

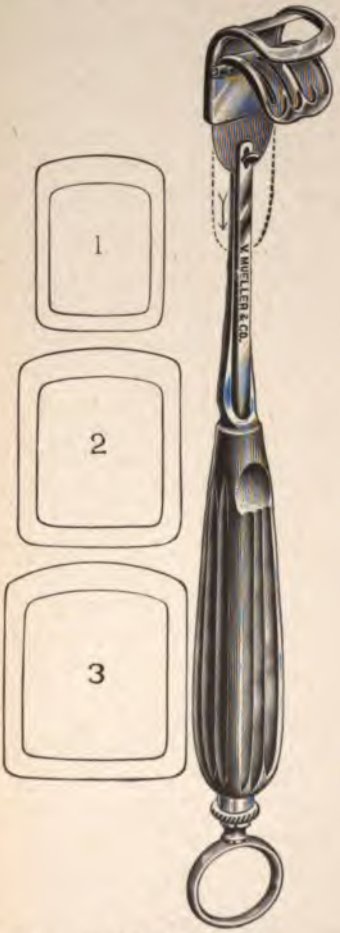

Fig. 149.—Collum's adenotome.

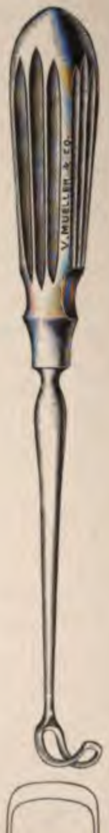

Fig. 150.—Barnhill's adenoid curette.

be inserted as far up and forward as is possible. To do this it is necessary that the handle touch the teeth of the lower jaw. The blade is then pushed home and the adenotome removed with the adenoid enclosed in the instrument. Following the use of the adenotome a Barnhill curette (Fig. 150) is inserted in the same manner and any remaining shreds of adenoid tissue are removed.

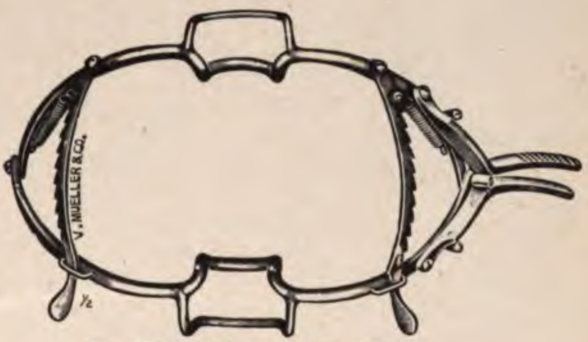

Fig. 151.—Whitehead's mouth gag.

Many operators prefer to remove the adenoid by means of a curette only. If the tonsils have been removed, the throat is sponged free of blood and the curette is introduced through the mouth into the nasopharynx, care being taken to carry it well up into the vault (Figs. 152 and 153). The handle is now made to describe the arc of a large circle, the fulcrum being the top of the fauces. The instrument should be pressed firmly and evenly to the vault and posterior wall during the upward movement of the handle (Fig. 154) of the curette.

The severed adenoid usually falls into the oropharynx when the curette is removed. The adenoid mass should be watched for and grasped with a forceps to prevent being accidently drawn into the larynx.

One or two sweeps of the curette are usually quite sufficient to remove all of the growth.

After the *removal of the instrument* the throat is again sponged free of blood and if necessary the patient turned on his right side or face to prevent the blood entering the trachea. The operator may now introduce the index finger of the right hand into the nasopharynx, to be certain that all of the adenoid tissue has been removed. The time required for the whole procedure need not be more than one to five minutes.

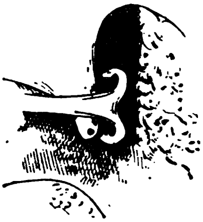

FIG. 152

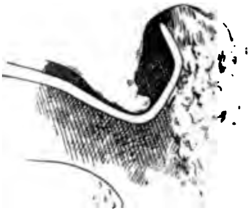

FIG. 153

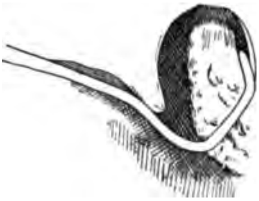

FIG. 154

After-treatment.—The patient should be kept on his side or face until he regains consciousness, to prevent the blood entering the esophagus and trachea. When he regains consciousness he should be directed to blow his nose in order to remove blood clots that may have lodged there.

He should be kept in bed for twenty-four hours following the operation. Local applications are usually not indicated.

Complications attending or following Tonsillectomy and Adenoidectomy.—1. There is danger of suffocation from the presence of blood in the trachea.

2. If the instrument is carried too far to the side of the pharynx, the orifice of the Eustachian tube may be injured, and a subsequent stricture result.

3. Secondary hemorrhage is rare.

4. The operation may be followed by infection of the middle ear and mastoid, and of the cellular tissue of the pharynx.

5. Dangerous primary hemorrhage may occur as a result of excessive fibrosis; or of the abnormal placement of the internal carotid and vertebral arteries. When dangerous hemorrhage takes place, it may be controlled by digital pressure; by pledgets of cotton saturated with a 10 per cent. solution of antipyrin; by eating crushed ice; by injections of normal horse serum and by keeping the patient absolutely quiet. Boettcher's adenoid hemostat is sometimes of service. If possible, the soft palate should be retracted; the bleeding-point found and grasped with an artery forcep and ligated; or a large sponge may be drawn up into the nasopharynx by a soft catheter passed through the nose. Bellocq's cannula is built on the correct principle but is usually too large to pass through a normal nose of a child. In severe hemorrhages the internal carotid artery may have to be ligated in the neck.

Thornwaldt's Disease. — **Definition.** — This is a condition in which there is a chronic inflammation of, and remnants of, partially atrophied *adenoids*. The thickening on the posterior wall of the nasopharynx is in the form of a ridge on either side of the median line. Pus accumulates in the groove thus created, becomes inspissated, and forms a central crust which is characteristic of this condition. It may be regarded as a postrhinitis sicca.

Treatment.—The treatment should be addressed to the attendant rhinitis; and the complete eradication of the fibrous adenoid ridges with the curet.

The Lingual Tonsils.—These are composed of masses of lymphoid tissue in the glosso-epiglottic space; or, as is commonly said, they are on the base or root of the tongue. They are slightly elevated nodules, some of which are pitted or concave on account of shallow follicles. There are present numerous mucous and seruminous glands, which are not found in "tonsils" elsewhere. The veins at the base of the tongue are very superficial, and are frequently the seat of varicosis.

Inflammations of the Lingual Tonsils.—The intimate connection between the lingual and the other tonsils causes them to respond to the same influences. Inflammation at the base of the tongue is rarely primary, but usually attends inflammation of the faucial tonsils. The same kinds of morbid processes occur in the lingual tonsils, namely, acute, subacute, and chronic inflammations. The crypts or crevices become filled with epithelial cells, leukocytes, and fibrinous exudate. They undergo hypertrophy as do their larger neighbors above. Deep and superficial abscesses occasionally form here also.

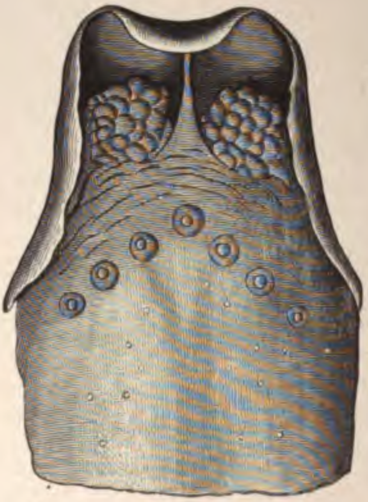

FIG. 155.—Hypertrophy of the lingual tonsils. (Cohen.)

Symptoms.—A sense of fulness and slight sticking or pricking pain upon deglutition and vocalization are the chief symptoms. Upon inspection with a laryngeal mirror the lymphoid nodules will be found to be enlarged and red; and if the crypts are involved, a yellowish material may be seen in them. Should there be an abscess of this region, the symptoms become quite marked; the pain is intense, the fever high, and deglutition of fluids difficult or impossible. Dyspnea may develop from obstruction to the glottic respiratory tract. The abscess may perforate the lingualis muscle and become or simulate a true *angina Ludovici.*

Prognosis.—*Catarrhal* and *lacunar* inflammations run their course within a few days or weeks; while the suppurative form may extend and give rise to serious symptoms. Edema of the glottis is rare. Should dyspnea of a dangerous nature occur, tracheotomy should be performed.

Treatment.—This should be much the same as for inflammation of the faucial tonsils. On account of the depression resulting from the dyspnea and difficult deglutition stimulants are especially indicated in the more acute or phlegmonous types. If an abscess is present, it should be opened with a curved tonsil bistoury. In making the incision care should be taken to avoid the lingual arteries.

Hypertrophy and Varix of the Lingual Tonsils.—The same *causes* that have already been given in reference to disease of the lymphatic tissue of the faucial tonsils hold true with the lingual tonsils. Oft-repeated inflammations of whatever source cause hypertrophy and varix of the lingual veins. The two conditions are usually associated; hence they will be considered together.

Hypertrophy and varix of the lingual tonsil is in the vast majority of cases a disease of adult life.

Symptoms.—The *subjective symptoms* may be classified as follows:

1. A feeling of fulness and irritation due to mechanical interference with the normal functions and movements of the parts. Patients describe the sensation in the glosso-epiglottic space as seeming to be caused by the presence of a bead, pea, straw, blister, or other foreign body. These signs disappear during deglutition, but quickly return.
2. Torticollis has been noted as an occasional sign.
3. The singing voice is impaired, not so much by the direct pathologic interference as by the inability properly to vocalize.

Continued attempts to do so cause the voice to become impaired. In other words, there would be no voice symptoms should the singer discontinue his special efforts in that direction.

4. Cough and hemming are prominent subjective symptoms. The effect is ineffective; the cough is of a brassy, croupy character.

5. Paroxysmal dyspnea is an occasional symptom.

The **objective** or **physical signs** as revealed by the laryngoscopic mirror are:

1. A unilateral or bilateral mulberry-like mass, which more or less obscures the epiglottis. In some cases the hypertrophy not being so marked, the tonsils appear as several small discrete nodules with varicose veins running between them.

2. The epiglottis is crowded backward, thereby obscuring the view of the larynx.

3. The mucosa of the larynx is usually hyperemic, while the cords are nearly normal.

4. Passive hemorrhages are often observed. The blood which has accumulated during the night is expectorated in the morning. This doubtless occurs during the daytime also; but as it is very slight, and the acts of swallowing and expectoration occur at frequent intervals, it is not noticed.

5. Faucial tenesmus, laryngeal spasm, and asthma have been reported as occasional symptoms of hypertrophy of the lingual tonsils.

Treatment.—Treatment offers a good prognosis. It should be both general and local.

The *local treatment* is palliative and surgical or semisurgical in character. The irritations, foul secretions, etc., which attend this and the other faucial, pharyngeal, and nasopharyngeal disease should be properly treated with antiseptic and emollient washes. The mineral astringents reduce the congestion, which is a prominent feature in the earlier stages of the disease. Lugol's solution gives good results.

℞	Potass. iod.	gr. xlviij
	Aquæ dest.	℥j—M.

Sig.—Apply with a cotton-mop to the lingual tonsils.

The sulphate of zinc, tannin, perchlorid of iron or aluminum are also useful in relieving congestion. Where there is pain or distress, a 50 per cent. solution of guaiacol in olive oil affords relief.

The *galvanocautery* is a good and safe method of removing the lymphoid masses or for destroying the varix. (1) The parts should be cocainized with a 10 to 20 per cent. cocain solution. (2) The patient should then protrude his tongue and grasp it with a napkin, as in laryngoscopy. (3) The operator introduces the laryngeal mirror with the left hand, while (4) the cautery electrode is brought to bear upon the lingual tonsil, or varicose veins. (5) The electric current should be connected and the (6) parts seared with the red-hot platinum point. If the veins only are to be treated, a small point should be used; while a flat electrode is more desirable for the treatment of the tonsil.

Specially devised tonsillotomes conforming to the curve of the dorsum of the tongue may be used. They act on the principle of the tonsillotome. One caution only will be given, namely, use the laryngoscope so as to avoid clipping the top of the epiglottis.

The operation is followed by complete relief from the distressing symptoms, but there will be some local pain and soreness for a few days.

The attendant disorders of the fauces, nose, and nasopharynx should also receive appropriate treatment.

Tuberculosis of the Pharynx.—**Pathology.**—This is a comparatively rare location for this disease and when present is usually secondary to pulmonary or laryngeal tuberculosis. A proper diagnosis is not always made, it being regarded as an acute pharyngitis. At first the membrane is red and infiltrated, and at a later period yellowish spots or miliary tubercles appear upon its surface. After a time they break down, the secretion becoming mucopurulent. The posterior pillars of the fauces become stiffened from infiltration, thereby permitting the entrance of liquid foods into the postnasal space. Scar tissue does not usually form in the wake of the ulcerative process, as only the superficial layers of the membrane are involved.

Symptoms.—Pain is a constant symptom that gradually increases from a dull ache to sharp, lancinating pains which are aggravated upon deglutition. The voice becomes thick and muffled. Cough is not more prominent than that due to the attendant pulmonary tuberculosis. The breath is fetid. The constitutional and local symptoms of pulmonary tuberculosis are present. Tubercular inflammation of the middle ear is often an extension of the process by way of the Eustachian tubes.

Prognosis.—This is quite grave, although not necessarily fatal. If the process in the pharynx is primary, the prognosis is much more favorable. Primary tuberculosis of the pharynx is, however, an extremely rare occurrence.

Treatment.—This is general and local and is identical with that given for tuberculosis of the larynx.

Syphilis of the Pharynx.—**Forms.**—It may manifest itself in four forms—*i. e.*, (*a*) primary, (*b*) secondary, (*c*) tertiary, and (*d*) congenital or hereditary.

The *primary form* is rare, as the fauces and pharynx are but slightly exposed, on account of their location, to primary inoculation. Chancre may be mistaken for tubercle and epithelioma, in both of which there is swelling of the lymphatic glands. It is usually recognized as syphilis only after constitutional symptoms develop.

The Secondary Form.—This occurs within one year after the primary infection.

Upon *inspection* a moist papule or a superficial erosion of the membrane may be seen.

Etiology.—This condition is still capable of infecting other individuals. Many children feeding from a wet-nurse thus contract the disease from the secondary manifestation on the mammæ. Such cases are usually regarded as inherited syphilis.

Pathology.—The pathology of this stage is as follows: (*a*) erythematous patches, superficial infiltration, exudative plaques, or condylomata on the pillars of the fauces, uvula, and velum.

Secondary Symptoms.—Subjectively they are those of an ordinary sore throat. A striking peculiarity of this stage is the symmetrical appearance of mucous patches and erythematous areas on the two sides. The history of the case, the peculiar skin eruption and the Wassermann test will enable the physician to make a correct diagnosis.

Tertiary Syphilis of the Pharynx.—*Pathology.*—This form appears at about the third to the fifth year after the primary infection, as a gummatous infiltration which rapidly breaks down, forming an ugly ragged ulcer. The pillars of the fauces are most often affected, the loose tissue affording a favorable field for round-cell infiltration. Another site often affected is in the median line, at the junction of the hard and soft palate. The ulcers may appear upon the tonsils, tongue, roof of the mouth, posterior wall of the pharynx or buccal surfaces. The deep variety of gumma is more circumscribed, and when it breaks down is more destructive. This form is responsible for most of the cicatricial bands remaining in the wake of syphilis of the fauces and pharynx.

Congenital Syphilis of the Pharynx.—*Occurrence.*—About 50 per cent. of the cases become manifest within the first five years of life. It occasionally appears in adult life.

Gross Pathology.—An ulcer appears with indurated edges. Its favorite site is the palate, although it may appear in any other situation. If the palate is the part involved, the nares may be invaded by direct extension through the floor of the nose. According to Lenox Browne: "The fauces, the nasopharynx, the posterior pharyngeal wall, the nasal fossæ, the septum nasi, the tongue, and finally the gums" are affected in order of frequency. In the secondary form the diseased areas tend to affect both sides symmetrically, while in this form the ulcer is almost always in the median line. It also has a predilection for the bony structures.

Symptoms.—They vary according to the extent and locality of the ulceration of necrosis. If the roof of the mouth is perforated, or if the pharyngeal muscles which shut off the pharynx from the nasopharynx are affected, the child cannot nurse or take the bottle. Coryza, excoriations, and scabs about the nostrils and upper lip may also be present. The child has what is popularly called the "snuffles."

Treatment of Syphilis of the Pharynx.—The *secondary stage* calls for the administration of mercury in mild doses, with especial care to avoid producing ptyalism. Direct the patient to wash the mouth thoroughly with a solution of the chlorate of potassium. The arsenic preparation known as salvarsan and neosalvarsan are exceedingly valuable in hastening the cure but should be supplemented by the usual treatment. Local applications of caustics, as acid nitrate of mercury, are of value in the active ulcerative stage. Iodin vapors or nebulæ exert a beneficial influence in those cases extending to the Eustachian tube and middle ear. Insufflation of iodol powder on the ulcerated spots has superseded the use of iodoform, as its odor is less offensive. The removal of enlarged tonsils should not be done during this stage, as the raw surface thus created would very likely assume the syphilitic form of ulceration.

The *tertiary stage* calls for the so-called mixed treatment combined with salvarsan or one of its equivalents. The iodid of potassium should be administered during the ulcerative stage.

It is wise to begin with about 3-grain doses and gradually increase them to the point of toleration. After the subsidence of the acute manifestations some form of mercury should be administered for about two years in order to prevent a recurrence of the destructive process.

Local applications of mineral caustics to the deep ulcers is to be recommended. The galvanocautery is, however, a better method. Gargles of the permanganat of potash, the chlorat of potash, etc., exert a favorable influence upon the morbid process, and keep the surfaces free of detritus, which would, if left *in situ*, excite irritation.

The *congenital form* should be treated about the same as the acquired. As children tolerate larger doses of mercury proportionately than adults, this remedy may be pushed more vigorously. The nose, mouth, and throat should be kept free from crusts by douches and sprays, after which the throat should be sprayed with oily solutions containing menthol, camphor, and eucalyptus.

A Comparison of Syphilitic and Tuberculous Ulcers (Lenox Browne).

Syphilitic Ulcers.	Tuberculous Ulcers.
Deeply excavated.	No apparent excavation.
Few granulations, highly inflammatory.	Much indolent granulation.
Deep red areola.	Faint areola.
Sharply cut edges.	Irregular and ill-defined edges.
Distinct demarcation.	Demarcation indistinct.
Yellow purulent secretion.	Grayish, ropy mucous secretion.
Discharge profuse.	Discharge scanty.
Penetrating to deeper tissues.	Superficial, with lateral in place of deep extension.
No fever.	High fever.
Slight pain.	Pain often severe.

Diphtheria.—**Definition.**—It is an acute infection attended by constitutional and local symptoms, due to a specific microörganism, known as the Klebs-Löffler bacillus. This is a rod-shaped bacillus varying in length from 1.5 to 6 micromillimeters, and in width from 0.5 to 0.8 micromillimeter. These bacilli are usually slightly curved and somewhat club-shaped at the ends. They are usually arranged in pairs, either parallel or at an angle to each other. They are sometimes single, and rarely end to end, forming long threads. They stain readily by Gram's method and Löffler's methylene blue.

Etiology.—1. The cause is the *Klebs-Löffler bacillus.* It was formerly thought that these bacilli remained localized in the fauces or other part of the body primarily affected; but they have been found in the blood, glands, intestines, and other organs of the body. They are, however, rarely found in any considerable number except at the point of initial infection. They find in the fauces, nose, etc., a suitable soil and temperature for their growth, and are propagated in great numbers. The toxins formed in consequence of the presence of the bacilli are absorbed and carried through the lymphatics and bloodvessels to every part of the system. In this way the various general symptoms are developed. The fever and great depression are due to the action of the toxins upon the nerve centres presiding over the production of heat and the processes of metabolism.

2. *Age* has an influence upon the susceptibility of the individual to the disease. While it may occur at any age, the majority of the cases are in persons under ten years.

3. *A cold, moist climate* is more favorable for its development than the warmer, or the extremely cold ones. The spring and fall of the northern temperate latitudes on this account are especially visited by this disease.

4. Certain *predisposing* and *systemic conditions* in a measure account for the prevalence of diphtheria in the cold, damp, temperate climates. For instance, catarrhal diseases, lymphoid hypertrophy in the fauces and nasopharyngeal space, are more prevalent in this sort of climate, and predispose to the diphtheritic infection.

The infection may be *transmitted* by railway trains, dust, drinking water, milk, domestic animals and fowls, and from one person to another.

Pathology.—The bacillus grows upon the mucous membrane and the toxins are elaborated and absorbed into the general circulation.

The first effect of the toxin is to produce a local edema and irregular temperature.

The *false membrane* is composed of three layers, namely: (*a*) the outer or superficial layer, which contains a network of fibrinous exudate, numerous bacilli, and a few leukocytes; (*b*) the middle layer, which contains a fibrinous exudate, many leukocytes, and few bacilli; (*c*) the inner layer, which is modified mucous membrane, the coagulated fibrin having penetrated the mucosa and replaced the surface epithelium.

Round-cell infiltration occurs in the mucous and submucous tissue. Streptococcus pyogenes, Staphylococcus aureus, and albus are also found in the inner layer. They cause secondary infection in the lymphatic glands, lungs, spleen, and kidneys. Inasmuch as the fibrin is deeply attached to the mucosa, its removal is difficult and is attended by hemorrhage.

The pseudomembrane due to cocci is feebly attached and easily removed, while the mucous membrane is red and shiny and does not bleed.

Bacteriologic Diagnosis.—This may be made by smearing a portion of the false membrane, or some of the mucopurulent secretion, upon a cover-glass, staining it with methylene blue and placing it upon a glass-slide and examining it with a microscope for the Klebs-Löffler bacillus. This method is rapid, but not certain to reveal the bacilli even when present in large numbers. A slower but much surer way is to make a culture of the mucus obtained from the diseased membrane on blood serum.

Clinical Diagnosis.—It is rare under one year of age. It is most often located on the faucial tonsils. In nasal diphtheria the discharge is fetid, sanious, and irritating to the upper lip, which becomes excoriated and encrusted. Epistaxis is a common occurrence in this type. The sloughing which causes the hemorrhage exposes a surface through which rapid infection may occur and cause great prostration, pulmonary edema, albuminuria, etc. The infection may gain entrance to the cranial cavity and cause meningitis. Nasal diphtheria is usually a mixed infection. The Eustachian tubes, middle ear, internal ear, and eye are sometimes the seat of diphtheritic pseudomembrane. Periostitis and edema over the mastoid are complications occasionally met with.

Inflammation of the cervical and submaxillary glands is an early sign in diphtheria. They may be swollen before the appearance of the membrane.

The diphtheritic membrane appears in varying colors according to the stage in which it is seen and the type of infection. It ranges from a white to a gray, yellowish-green, brown, and even to black, according to the quantity of pus and blood mixed with it.

The *Schick test* to determine the degree of individual immunity has recently been perfected and is especially valuable in institutional work. Some persons possess a natural immunity to diphtheria and in these the prophylactic use of antitoxin is unnecessary.

Symptoms.—They vary with the severity of the disease, type, and location of the area of infection, age of patient, and the stage of the disease.

The *period of incubation* ranges from two to ten days. Symptoms are not always present during this period; but when present they consist of general malaise and slight chilliness.

At the *end of the incubation period* the temperature ranges from 99° to 103° F. It usually remains about the neighborhood of 100° F. Great pyrexia is not a characteristic feature of diphtheria.

The onset of the disease is attended by slight elevation of temperature, quickened pulse, slight chills or chilly sensations, dry throat, discomfort or pain upon deglutition, malaise, stiff neck, aching in bones, headache, and other febrile phenomena. Any or all these symptoms may be very slight or wanting. By the end of the first day or beginning of the second there are lumbar and dorsal pains, the skin is flushed, the pulse quick and perhaps feeble, the cervical glands enlarged and tender, the urine scanty and high colored, the patient dull and unobservant.

The *pseudomembrane* at the beginning of the *first day* appears as a delicate pearly gray film, slightly elevated above the surface of the mucosa, which is swollen and dark red in color. If the diphtheritic membrane is limited to one side, the catarrhal turgescence of the mucosa will also be unilateral.

At the beginning of the *second day* in untreated cases the membrane, which on the first was in patches, is now extended until it covers all or nearly all of the tonsil. Its color is yellowish and opaque, and the surface presents a soft, velvety appearance. It stands out more prominently from the surface of the mucosa, giving the impression of considerable thickness. Mucopurulent secretion is noted about the edges, separation from the underlying mucosa having begun. It may come away in fragments or *en masse*.

By the *third day* in untreated cases the membrane is brownish or blue in color owing to the admixture of blood. It is more elevated from the surface and separation is complete. The temperature subsides, and if it should become elevated again, reinfection has occurred or a new area has become involved.

Invasion of the larynx in untreated cases is marked by hoarseness or aphonia, which is followed some hours later by dyspnea, both inspiratory and expiratory, although inspiration is more difficult. Subclavicular and abdominal depression are marked. The laryngeal invasion is attended by a sudden increase in the temperature. The membrane may be expelled in part or as a cast on the third or fourth day, and the process of resolution or that of the reformation of the pseudomembrane may now take place.

Treatment.—The treatment may be studied under the following divisions: (1) prophylactic, (2) general or systemic, (3) local, and (4) surgical.

Prophylactic.—Isolation should be enforced until the cultures no longer show the Klebs-Löffler bacillus. Where strict surveillance and hygiene is not possible, immunizing doses of antitoxin should be given to those exposed.

General or Systemic Treatment.—The antitoxin treatment should be given at the earliest possible moment, as it is most effective when administered during the early stages of the disease. It is preferably given by hypodermic injections.

The *quantity necessary* varies with the individual, the epidemic, the part infected, and the age of the patient. In a general way it may be stated that the dose for a child under five years of age should be from 1500 to 3000 units; while 3000 to 10,000 units should be given to patients more than five years old. A prophylactic dose is from 600 to 800 units. The dose may be repeated in twelve to twenty-four hours if improvement has not set in. A third dose may also be given without danger in unyielding cases. The effect of the antitoxin is manifested by the subsidence of the toxic symptoms and the disappearance of the pseudomembrane.

Among the *remedies* which promote the elimination of the toxin, calomel stands at the head. It should be administered in full doses at frequent intervals so as rapidly to obtain its effect. Turpentine in milk has advocates, and it will at least stimulate the excretions through the kidneys. The tincture of iron in glycerin (1 dram to the ounce), given in teaspoonful doses every two hours, has a decided influence upon the local as well as upon the general condition of the patient. It adds to the cardiac, respiratory, and blood power, enabling the system more effectually to resist the toxins already in the circulation, and perhaps to check their production.

Local Treatment.—Local applications of *lactic acid* in one-fourth to one-half strength to the diphtheritic membrane by means of a cotton-wound applicator facilitate the exfoliation of the pseudomembrane from the mucosa.

The *normal salt solution* (0.3 per cent.) or ***boric acid*** in solution may be used every hour in the mouth, nose, and fauces by means of a syringe. This is preferable to the use of gargles, as they are usually inefficient in children. This is especially true in diphtheria attended by marked depression.

The following formula prescribed by Löffler is a ***local remedy*** of considerable value:

℞.—Menthol		10	parts
Toluol		26	"
Sol. perchlorid of iron, fort.		4	"
Absolute alcohol	q. s. ad	100	" —M.

Sig.—To be applied locally once or twice daily.

Inhalations of the vapor of slaked lime is an old and valuable remedy in laryngeal diphtheria. It should be administered as follows: A temporary wigwam or cone-shaped tent should be constructed by the use of sheets. A chair, and a wooden vessel of two or three gallons' capacity half-filled with water, should be placed within the tent. Two or three pounds of fresh lime should then be thrown into the vessel, where it soon throws off steam charged with small particles of the lime. The patient should be placed upon the chair or held upon the lap of the nurse within the wigwam for five to ten minutes. This procedure should be repeated every two or three hours. In this way the diphtheritic membrane may be caused to separate from the trachea and bronchial tubes and be expelled in fragments or *en masse*. Turpentine may be added to the lime vapors with advantage.

Antiseptic alkaline sprays are of value in mild cases. They should be used in the fauces every hour.

Surgical Treatment.—*Indications.*—When there is progressive asphyxia, signalized by aphonia, dyspnea, stridor, and recession of the chest and abdominal walls either ***intubation*** or *tracheotomy* should be done without delay.

The *dangers* attending either of the procedures is so small as compared with those of non-interference that the latter should not be allowed to weigh against the former.

Intubation.—Intubation after the method of O'Dwyer has largely superseded tracheotomy, the latter being used only in emergency and where the membrane has formed in the lower tracheal tract.

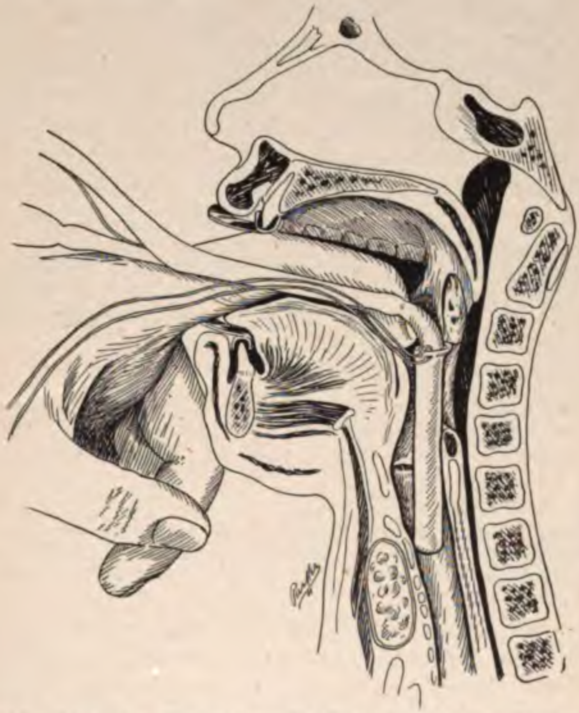

FIG. 156.—The tube passing through the chink of the glottis, the index finger still holding the epiglottis against the base of the tongue. A stout loop of thread is attached to the tube to provide for its speedy removal in case suffocative symptoms follow its introduction, and in case it is accidently engaged in the esophagus.

Method of Introducing the Tube.—The patient should be taken on the lap of the nurse, his head held firmly against her left shoulder with the left hand while his hands and arms, folded in front, are held with her right hand and arm. If need be, a binder or sheet may be wound about the child, should be suddenly elevated and slightly drawn forward, the tube being made thereby to pass *downward and forward* along the palmar and thumb surface of the finger into the larynx. If the handle is not thus suddenly elevated and drawn forward, the tube will enter the esophagus.

As the patient is in great distress the whole procedure should be done with precision and speed.

Before introducing the tube it should be threaded through its shoulder by a strong silk or linen thread about sixteen inches long, the ends of which are tied, forming a loop. This should be hooked over the little finger of the left hand ready for use should the tube enter the esophagus, or need to be removed for any other reason.

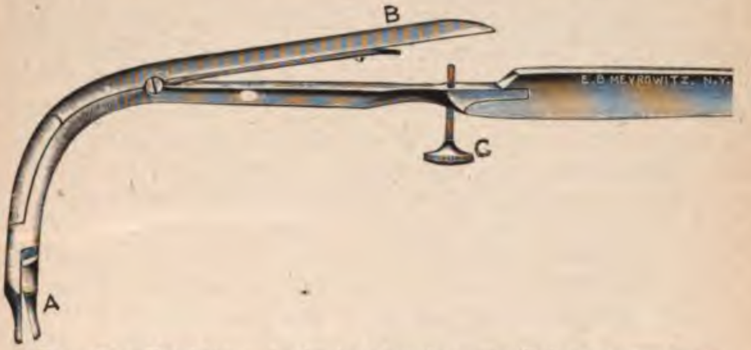

Fig. 158.—Extubator: *A*, the jaws partly open; *B*, lever, pressure on which in a downward direction opens the jaws; *C*, screw which regulates the extent to which the lever may be depressed and the jaws opened.

When the tube is properly adjusted, the thread may be cut and removed, the index finger being introduced and held against the shoulder of the tube to prevent its withdrawal, or the thread may be left attached, looping it over the ear and securing it to the cheek with adhesive plaster. The objection to this method is that the child may pull on the string and thus remove the tube.

If the tube is too small, it will be coughed up.

The *objections to intubation* are: (*a*) the liability to cough up the tube and the necessity of calling the physician to reintroduce it; (*b*) it sometimes interferes with deglutition: liquid food can usually be swallowed. If food enters the trachea, the patient should be made to lie upon the stomach and take the food through a tube; (*c*) the opening of the tube being small, it sometimes becomes obstructed by false membrane, thereby necessitating its immediate removal. This is sometimes accomplished by the violent efforts of the child.

The *tube should be left in place* until the disappearance of the faucial membrane, the great depression, and the feeble, rapid pulse.

Its *removal* is accomplished with the extubator shown in Fig. 158. The tip should be introduced into the mouth of the tube, guided by the index finger of the left hand. It is then widened by pressure upon the lever *B*, and the tube removed by reversing the movements used in its introduction.

Tracheotomy.—*Technic.*—High tracheotomy is preferable to the low tracheotomy of Trousseau, as the bleeding in the latter is more profuse.

The cricoid cartilage is located with fingers of the left hand and while the larynx is held firmly between the thumb and the second finger the skin and subcutaneous tissue is incised in the median line beginning with the cricoid cartilage and carrying it downward ½ or 1 inch. If time permits, all bleeding-points should be checked with artery forceps or ligatures. The two or three upper rings of the trachea are now incised with a knife. The tracheal aperture is held open with retractors and the cannula inserted. The cannula is secured in position by pieces of tape passed around the neck.

After-treatment.—The wound about the tracheotomy tube should be covered with antiseptic gauze. The opening in the tube should be lightly covered with one or two thicknesses of gauze to filter the dust from the inspired air. The atmosphere of the room should be kept moist by steam vapors to provide moisture for the inspired air. The temperature of the room should be maintained *constantly* at about 70° F. The inner tube should be removed and cleansed by the nurse every two hours. The entire tracheotomy tube should be removed every twenty-four to forty-eight hours and another of the same size introduced in its place. Warm antiseptic sprays may be used from time to time through the tubes to thin the mucous accumulations in the trachea.

MALFORMATIONS OF THE UVULA.

Varieties.—Bifid Uvula; Complete or Partial Absence of the Uvula.

Symptoms.—Cough and irritation in the pharynx sometimes occur from the presence of a bifid uvula.

Treatment.—The treatment consists of removing the mucous membrane on the opposed surfaces of the divided uvula and bringing them together with catgut sutures.

DISEASES OF THE UVULA.

Ulceration of the Uvula.—**Occurrence.**—This is rare as a primary condition, and is characterized by pain upon deglutition. The ulcer is usually upon the posterior surface, and can only be seen with the aid of the laryngeal mirror. It may be the cause of a supposed epipharyngitis.

Treatment.—The treatment should consist of the application of the solid stick of the nitrate of silver or the actual cautery.

Mycosis of the Uvula (*Leptothrix*).—Here, as in tonsillar mycosis, there are the ivory-colored, pointed, and elevated patches of leptothrix.

Acute Inflammation, or Edema of the Uvula.—**Etiology.**—The etiology is very similar to that of acute tonsillitis.

Symptoms.—The symptoms are those of slight obstruction, pain upon swallowing, and a tickling cough. A general pharyngitis is often associated with it.

Treatment.—During the acute stage it should consist of scarifications and alum or zinc sulphate washes. After the subsidence of the acute stage it may be necessary to amputate the uvula on account of the elongation. When thus elongated it gives rise to a distressing dry cough.

Elongated or Relaxed Uvula.—This condition constitutes the major portion of the affections of the uvula requiring treatment.

Etiology.—It is commonly associated with inflammation of the pharynx, and more especially in those who strain the voice in singing, speaking, or coughing.

In its enlargement the vascular, glandular, fibrous, or muscular tissue may predominate.

There is sometimes an entire absence of muscular fibers in the elongated uvula.

Symptoms.—Coughing paroxysms frequently occur. The cough is dry and usually more pronounced in the morning. There is a tickling sensation in the throat with more or less discomfort and pain. The expectoration is occasionally streaked with blood, which alarms the patient, as he suspects tuberculosis.

Singers are particularly annoyed by this condition, as it incapacitates them from pursuing their profession. The special symptoms in singers are pain and discomfort after using the voice, loss of brilliancy, uncertain register, impaired range, and unsteadiness of tone. In time hoarseness supervenes, thereby completely destroying the singing voice.

Treatment.—The classical treatment has long been astringent gargles of alum water and solutions of tannin. Astringent lozenges with eucalyptus, cubebs, chlorate of potash, guaiacum, and menthol are now more commonly employed. After using one or more of these remedies for a few days the amount of permanent elongation can be accurately estimated. If the elongation is still excessive, a portion of the uvula should be amputated. The incision should be made in such a manner as to leave the wounded surface toward the posterior wall of the pharynx. The act of swallowing food will thus be more free from discomfort and irritation. The operation may be performed by grasping the tip of the uvula with a pair of toothed forceps, drawing it forward and downward and amputating above the forceps with curved uvula scissors; or it may be done with a iuvulatome, an instrument resembling Mathieu's tonsillotome; it may also be removed by means of a snare. A 5 per cent. solution of cocain should be applied to the surface at intervals for a few minutes before resorting to either of the foregoing procedures.

NEOPLASMS OF THE PHARYNX.

Fibroma of the Nasopharynx.—**Etiology.**—This is a firm fibrous growth frequently arising from the base of the sphenoid bone and spreading to adjacent tissues and cavities, more especially the nose. It springs from apparently normal tissue; and occurs in persons who are otherwise healthy. Males seem to monopolize the disease, but few cases occurring in females. Age exerts a decided influence, it being rare under ten and over twenty-five years. Puberty marks the commencement of most cases. The growth is spontaneously arrested at about the age of twenty to twenty-five years.

Symptoms.—The early sumptoms are those of slight irritation and nasal stenosis. As it progresses these become more pronounced. The extension and development of the tumor are usually rather slow, but may be quite rapid. Headache and aprosexia are usually present, as in all types of nasal stenosis. Frog-face or a wide nasal bridge becomes a prominent symptom. As the neighboring cavities are invaded, pain becomes a symptom of importance. Hemorrhage may become a grave complication. The surface of the growth is interlaced by thin-walled vessels, which probably account for the hemorrhage. Upon examination with the rhinoscopic mirror a smooth, rounded tumor of a pale pinkish color is seen. Sometimes it is of a deep red or purplish color.

Diagnosis.—A fibroma is firm, dense, and elastic to the touch, while a sarcoma is soft and yielding. Both bleed upon slight mechanical irritation. The pain in sarcoma is apt to be referred to the ear. The fibroma may be differentiated from fibropolypus by its firm, broad attachment, while the fibropolypus is pendulous and easily moved. The fibropolypus is also much softer to the touch.

Prognosis.—As there is no limit to the growth, it may invade vital parts and become quite a grave disease. The tendency to severe hemorrhage also adds to the gravity of the condition by impairing the health or causing death. If it is early recognized and properly treated, the prognosis is quite favorable.

Treatment.—Small growths, if somewhat pedunculated, may be removed by means of a heavy snare or forceps, either through the nose or mouth. Larger growths, which have extensive inflammatory adhesions to the adjacent structures, may require an external or more radical operation. As the hemorrhage is usually profuse, the operator should be prepared to ligate the external carotid. *Injections* of the chlorid of zinc in saturated aqueous solution, repeated at intervals of two or three days for a fortnight or more, have occasionally been successful. Acetic acid has been used in the same manner.

Fibrous Polypus of the Nasopharynx.—Occurrence and Structure.—This is more rare than fibroma. It starts in the vault of the pharynx and, like fibroma, extends into the nose. The *pharyngeal* portion is more *fibrous*, while the *nasal* extension is more *myxomatous*, in structure. Unlike fibroma, it does not distort the neighboring bones and cavities, nor is it attended by hemorrhage. About two-thirds of the cases occur in females between the fifteenth and thirtieth years.

Prognosis.—The prognosis is quite favorable under treatment. It is less apt to recur after removal than simple polypus.

Diagnosis.—Articulation is impaired, as the uvula and velum lose their mobility. The tumor hangs free in the nasopharynx and may sometimes be seen projecting below the soft palate.

Treatment.—The growth may be seized through the mouth with a strong pair of forceps and twisted from its attachments. Damage, however, may be done to other structures by this procedure. If it is attached to the mucosa and periosteum of the body of the sphenoid, the bony walls of which are sometimes quite thin, evulsion may fracture the thin plate of bone and expose the sphenoidal sinus.

The *snare* is a safer instrument to use in these cases. Some ingenuity may be required to engage the growth in the loop.

Chondroma of the Nasopharynx.—**Symptoms.**—These are much the same as those of fibroma, except that the hemorrhage is absent. Nasal stenosis, aprosexia, headaches of long duration, and syncope may occur during the progress of the disease. Great distortion of neighboring bones and cavities occurs, as in fibromata. To the touch the tumor offers a firm, hard, resisting surface. It is not so hard but that a needle may be thrust into it. With the postrhinoscopic mirror it appears as a smooth, whitish-pink tumor.

Treatment.—Nothing short of a radical surgical operation will be effective. Langenbeck's operation for the temporary removal of the superior maxilla is the best method of exposing the growth.

Sarcoma of the Nasopharynx.—Unlike sarcoma in other parts of the body, it seems to occur with almost equal frequency at any time between the ages of one to fifty years. It occurs most frequently in males.

Symptoms.—They are about the same as those of fibroma, except that the hemorrhages are not so serious. There is an offensive discharge which is probably septic and accounts for the early disturbance of the general health. As the disease advances there are difficult deglutition, dyspnea, pain in the ear, impaired hearing or complete loss of hearing. Objectively the tumor is soft and elastic, bleeds easily upon digital examination, unlike fibropolypus. The glands of the neck are not enlarged as they would be in carcinoma. Sight may be destroyed by pressure upon the optic nerve in its passage through the sphenoid, but both it and hearing may return after the removal of the tumor. There is a tendency to recurrence after removal.

Treatment.—The treatment should be about as for fibroma and chondroma.

Carcinoma of the Nasopharynx.—**Symptoms.**—In its early stage it gives rise to the same clinical phenomena as sarcoma. There is early glandular enlargement, which persists throughout the progress of the disease.

Treatment.—Early diagnosis and complete removal offer the only hope. Radium has been used with indifferent success.

NEOPLASMS OF THE FAUCES.

Varieties of Benign Tumors.—On account of the character of the histologic formation of the fauces benign neoplasms are not often found here.

Papilloma.—Papilloma of the uvula, soft palate, or faucial pillars is the most common form of neoplasm seen in this region. These growths appear as warty vegetations and show little disposition to extend. Occasionally they grow quite rapidly. As they present the usual appearance of papillomata growing upon mucous membranes generally, an extended description of them will not be given here.

Treatment.—This consists in removal with a snare, scissors, or knife. A portion of the healthy mucous membrane should also be removed and the point cauterized to prevent a return of the growth.

Fibroma of the Soft Palate.—It develops rapidly and has no other symptoms than those due to mechanical interference. If operated upon early, there seems to be no danger attending removal.

Angioma of the Fauces.—Several cases have been reported. It consists of a network of small bloodvessels with very thin walls. The surface of the tumor is quite sensitive and gives rise to considerable pain upon deglutition.

Treatment.—The treatment consists of radical removal with the cautery loop, as the hemorrhage would otherwise be severe.

Malignant Tumors of the Tonsils.—These are comparatively rare affections.

The **varieties** found may be thus classified:

EPITHELIOMATA.

1. Squamous.
2. Alveolar.
3. Columnar.

SARCOMATA.

1. Round cell.
2. Spindle cell.
3. Lymphosarcomata.
4. Angiosarcomata.

Occurrence.—Sarcoma of the tonsil seems to be a disease of adult life, whereas in other parts of the body it is peculiar to the earlier periods. Epitheliomata belong to the later periods of life. They are found most often in males, although the difference is not marked enough to make this distinction of practical importance.

Etiology.—The location and function of the tonsils and fauces, and the irritation to which they are subjected in a measure account for the great frequency with which they are affected by malignant disease. The low resisting power of all lymphoid tissue must also be taken into account. The use of alcohol and tobacco seems slightly to predispose to malignant disease of the fauces.

Pathology.—The pathology of malignant tumors of the fauces and tonsils differs in no way from such morbid processes elsewhere in the body; hence will not be described in detail in this work.

Symptoms.—The early symptoms of sarcoma and epithelioma are those due to mechanical interference; a little later there is the addition of sharp, lancinating pain.

Prognosis.—Sarcomata affecting the *mucosa* may not be a very grave affection. Under early operative interference a cure may be expected. When, however, it attacks the substance of the tonsil, it becomes a grave disease, and the prognosis should be guarded. The tonsil, being a part of the lymphatic system, makes the disease much more liable to extend to contiguous parts.

Epithelioma of the tonsil or fauces is grave in the extreme. It is so located as to favor its rapid spread to adjacent lymphatic glands.

Treatment.—Operative measures offer the best hope. These can usually be done through the mouth; hence no special description of the primary incisions through the skin and underlying tissues will be given. In some cases a preliminary tracheotomy should be given. This question must be decided in each individual case according to the symptoms. The carotid artery may be ligated as a preliminary step; otherwise severe hemorrhage may occur. Should it be necessary to gain entrance into the pharynx through the lateral wall, the choice will lie between a lateral pharyngotomy and a subhyoidean pharyngotomy.

Radium should be used as a postoperative measure.

THE LARYNX.

EXAMINATION OF THE LARYNX.

Good illumination and the proper instruments are essential to the examination of the larynx.

The *means of illumination* will of necessity depend very much upon the environment of the physician. If, for example, he is located in the country, where gas and electricity are not to be had, he must depend upon sunlight, the oil lamp, or the portable electric lights.

Sunlight furnishes an ideal illumination, but for obvious reasons is not always available. An ordinary *oil lamp* is always available in most country places and is quite satisfactory.

In the larger cities, however, where gas and electricity are in almost every home and office building, their use is more practicable, and with the proper appliances furnish a more brilliant illumination.

The ordinary flat gas flame, the argand burner, or the hooded Welsbach gas burner may be used. The Welsbach burner makes an ideal light, as it is both brilliant and steady. *Gasoline* is now utilized, by means of a very simple contrivance, to furnish gas for the Welsbach burner, thus making it possible for the practitioner in the most remote places to have an ideal mode of illumination.

Electricity is, however, the light *par excellence* for the specialist, as it is cleanly, and throws out less heat, which is a desideratum of no small importance where the lamp or gas-light may be in constant use for hours at a time.

Instruments.—If sunlight, the oil lamps, gas, or the electric incandescent globe are utilized for the source of illumination, it is necessary to use a concave head mirror to throw the rays of light into the oral cavity. There are electric head lamps which do away with the necessity of using a mirror reflector. The Phillips and the Kirstein head lamp are perhaps the best on the market. The Kirstein is better adapted for ear work, as it throws the rays of light in a direct line with that of vision.

In addition, a napkin for holding the tongue, a tongue depressor, and three or four laryngeal mirrors of various sizes should be at hand for making the examination. The larger sized ones are generally preferable for getting the laryngoscopic image.

Method of Examination by Indirect Laryngoscopy.—The patient is placed on a revolving chair a little to the front and left side of the light, and at such a height that his mouth is about on a level with the light. He is instructed as to what is to be done, and how he can aid in doing it.

After having adjusted the light about nine inches to the right and a little back of the patient's ear, the patient is told to protrude his tongue, the end of which is seized gently, but firmly, between the folds of a napkin (Fig. 159), and pulled gently forward and downward, using the index finger beneath the tongue as a fulcrum. This simple precaution avoids giving the patient pain by dragging the tongue down over the teeth. The patient is directed to relax the lower jaw and tongue, and breathe normally. While he is in the act of doing this, a large warm laryngeal mirror is inserted, holding it as a pen, keeping the back of it well up toward the roof of the mouth, following the curve of the vault back to the uvula and soft palate, which is lifted gently, but firmly, if need be, against the upper and posterior wall of the pharynx. Especial care should be exerted to avoid touching the tongue upon its posterior aspect, as violent retching may be thus produced.

The face of the mirror should now look forward and downward, and the light from the head mirror or lamp be directed upon it. The rays will thus be reflected downward upon the larynx, and the image returned to the eye. The examination should be done quickly and skilfully, as the parts will not long tolerate the presence of the mirror. Should retching or coughing be excited the examination should be discontinued at once,

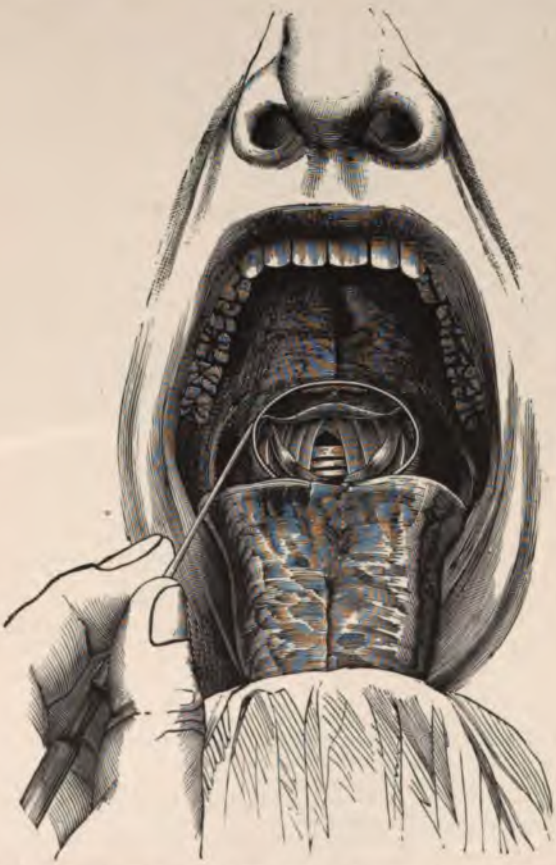

FIG. 159.—Laryngeal mirror in position, displaying the laryngeal image. (Cohen.)

and the patient's mind diverted from his throat. A few sips of ice-water will usually allay the irritability of the parts, and the examination may be repeated.

The position and action of the vocal cords and arytenoid cartilages may be brought into view by directing the patient to utter the syllables "eh," "ee," and to take a long, deep breath.

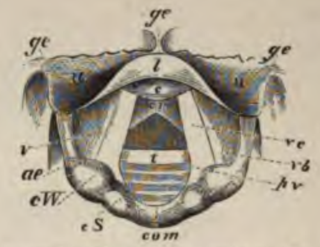

FIG. 160.—Laryngoscopic diagram, showing the vocal cords widely drawn apart, and the position of the various parts above and below the glottis, during quiet breathing (from Mackenzie): *ge*, glosso-epiglottic folds; *u*, upper surface of epiglottis; *l*, lip or arch of epiglottis; *c*, protuberance of epiglottis; *v*, ventricle of the larynx; *ae*, aryepiglottic fold; *cW*, cartilage of Wrisberg; *cS*, cartilage of Santorini; *com*, arytenoid commissure; *vc*, vocal cord; *vb*, ventricular band; *pv*, processus vocalis; *cr*, cricoid cartilage; *t*, rings of trachea. (Seiler.)

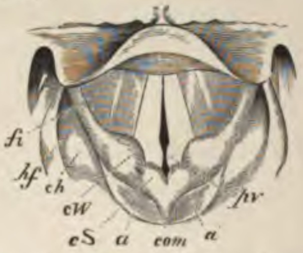

FIG. 161.—Laryngoscopic diagram, showing the approximation of the vocal cords and arytenoid cartilages, and the position of the various parts, during vocalization (from Mackenzie): *fi*, fossa innominata; *hf*, hyoid fossa; *ch*, cornu of hyoid bone; *cW*, cartilage of Wrisberg; *cS*, cartilage of Santorini; *a*, arytenoid cartilages; *com*, arytenoid commissure; *pv*, processus vocalis and cartilages of Seiler. (Seiler.)

The Laryngoscopic Image.—The image is an inverted one, as it is only the reflection from the mirror that strikes the retina. To remember this is of importance in making topical applications and in operating upon the larynx. The movements of the instruments thus used will be the reverse of that which seems natural, and considerable experience will be required to accustom the operator to them. The beginner should provide himself with a model mouth and larynx, or construct one of a piece of large tubing, and practice upon it with the laryngeal mirror, reflected light, and curved laryngeal instruments until he is accustomed to the reverse movements reflected from the laryngeal mirror.

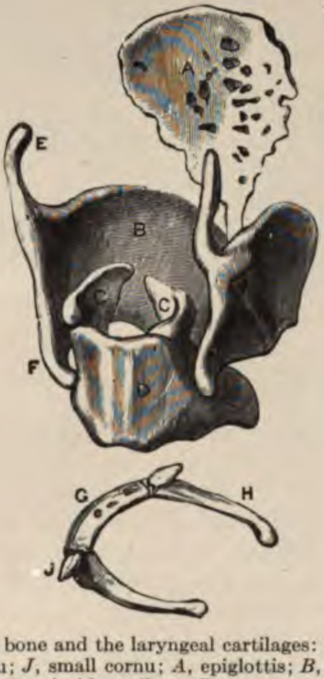

Fig. 162.—Hyoid bone and the laryngeal cartilages: *G*, body of the hyoid bone; *H*, large cornu; *J*, small cornu; *A*, epiglottis; *B*, thyroid cartilage; *C*, arytenoid cartilage; *D*, cricoid cartilage; *E*, upper cornu; *F*, lower cornu of the thyroid cartilage. (Ellis.)

Direct Laryngoscopy.—This is best accomplished by means of the Killian suspension instrument which is an adjustable gallows-like arrangement whereby the head of the patient (dorsal position) is suspended over the end of the table with no means of support other than the tongue spatula which is swung from the horizontal bar of the apparatus. Consequently the patient's head is supported by the structures attached to the hyoid bone and the lower jaw.

The advantages of direct laryngoscopy by means of the Killian apparatus are: (1) A direct view of the larynx is continually present. (2) The operator works in an easy position with both hands free. (3) The danger of inspiration of blood or pieces of tissue is lessened. (4) The patient suffers but little discomfort and can tolerate prolonged examinations or operations within the larynx.

The more common indications for its use are: (1) Diagnosis. (2) Removal of foreign bodies from the larynx or mouth of the esophagus. (3) Currettage or treatment of the larynx. (4) Removal of intralaryngeal growths and hypertrophies.

Laryngoscopic Landmarks.—The general anatomy of the cartilages and muscles is shown in Figs. 162, 163, and 164.

The laryngoscopic mirror will show the following landmarks.

The **epiglottis** with its crescentic or V-shaped edge standing up against the base of the tongue. It occupies the upper portion of the mirror and presents its concave surface downward.

It should be *examined* for deformities, neoplasms, catarrhal inflammation, and tubercular, syphilitic, and malignant infiltrations. Its color should be noted as should also the condition of the veins and arteries upon its surface.

The next landmarks to engage the attention are the *rounded prominences* in the lower field of the mirror. They are covered with mucous membrane and mark the location of the **arytenoid cartilages.** They play a prominent part in vocalization and are often the seat of tubercular infiltration. Their color, size, and symmetry should be noted. They may be much enlarged from tubercular infiltration or broken down by ulceration. One side alone may be thus affected.

The **vocal cords** appear as pale, pinkish bands, occupying the central portion of the mirror in its perpendicular axis. They seem to unite in the upper portion of the mirror and to spread apart as they pass into the lower field. A reversal of the image gives the true position of the parts, namely: the cords unite anteriorly at the base of the epiglottis, and

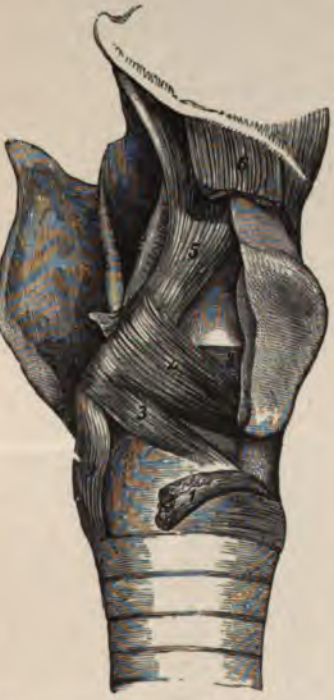

Fig. 163.—View of the internal muscles of the larynx: *1*, cricothyroideus detached; *2*, crico-arytenoideus posticus; *3*, crico-arytenoideus lateralis; *4*, thyro-arytenoideus, superficial part; *5*, depressor of the epiglottis; *6*, thyrohyoideus, cut; *8*, deeper part of thyro-arytenoideus. (Ellis.)

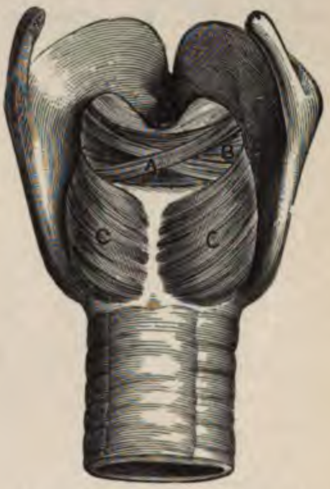

Fig. 164.—Posterior view of the larynx: *A*, superficial part of the arytenoideus muscle; *B*, deep part of the arytenoideus; *C*, crico-arytenoideus posticus. (Ellis.)

pass backward to the arytenoid cartilages. During deep inspiration the cartilages separate, and thus cause the cords to spread apart in the lower field of the mirror.

To the outer side of either cord may be seen a band or fold of mucous membrane known as the **ventricular band,** pocket ligament, or false cord.

The ventricular bands form the superior boundaries of the **ventricular pouches** or pockets.

Both the bands and pouches may be the seat of catarrhal inflammation, edema, and glandular enlargement.

MALFORMATIONS OF THE LARYNX.

They may be **congenital** or **acquired.**

Congenital Malformations.—**Varieties.**—They may be grouped under three headings, namely:

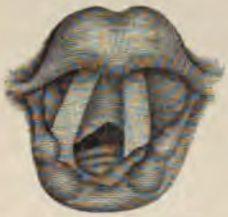

FIG. 165.—Laryngeal stenosis due to web between the vocal cords. (Cohen.)

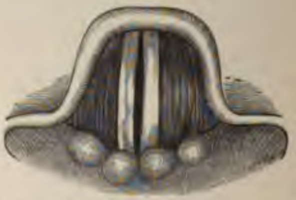

FIG. 166.—Faulty approximation of the vocal cords as often observed in chronic hypertrophic laryngitis. (Coakley.)

(*a*) *Congenital stenosis,* which is usually due to a *web* (Fig. 165) stretched across the anterior commissure from cord to cord, or from the ventricular bands. This condition may be present for years without attracting notice, when some intercurrent disease of the larynx brings it under the observation of the physician. The web is usually of the same color as the cords.

(*b*) Another form of congenital malformation is *laryngeal dilatation*, or *laryngocele*. This is rare in man, but is quite common in some of the lower animals.

(*c*) *True hypertrophy,* or enlargement of the normal tissue, is occasionally found as a congenital condition, although it is usually acquired.

Treatment.—If the web causes stenosis of the larynx, it may be treated by the introduction of an O'Dwyer tube, leaving it in place for a few hours daily. It may be necessary first to cut the web with a cutting dilator.

If there is stenosis from *true hypertrophy*, the enlarged mass should be removed with instruments or caustic applications, as fused crystals of chromic acid, or with the actual cautery. Chemical caustics are to be cautiously applied, as they are apt to excite the formation of cicatricial bands. If the hypertrophy is large enough, it may be removed with the biting laryngeal forceps.

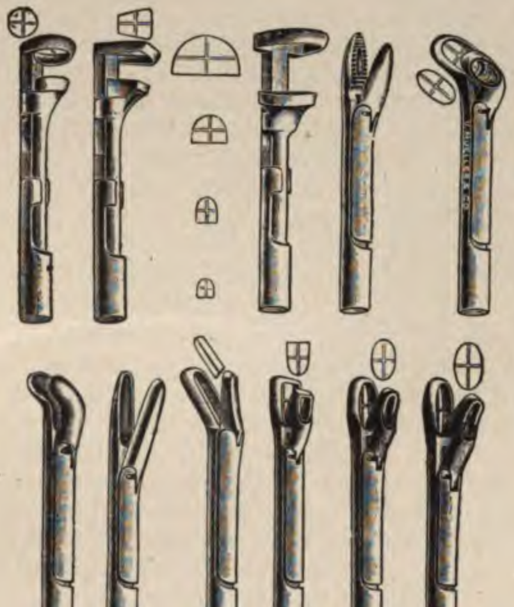

FIG. 167.—Universal cutting and grasping tips for laryngeal instruments.

Acquired Malformations.—**Etiology.**—They are usually due to some injury, as: attempted suicide by cutting the throat; the inhalation of a corrosive vapor; or the deglutition of scalding or caustic fluids. Syphilis, lupus, tuberculosis, cancer, and benign neoplasms also give rise to malformations.

Syphilitic Stenosis of the Larynx.—*Etiology.*—The stenosis may be due to edema, or to cicatricial bands and webs stretching from cord to cord, or between the ventricular bands. Edema may occur at any period in the course of the disease.

Symptoms.—(*a*) Those due to interference with voice production, as permanent hoarseness and restricted range of register; (*b*) those due to interference with respiration, as repeated attacks of dyspnea. Such attacks recurring at frequent intervals for a number of years are very suggestive of syphilitic disease of the larynx, (*c*) cough, (*d*) pain, (*e*) dysphagia, and (*f*) expectoration are often present. The expectoration is, however, usually scanty.

Treatment.—The usual constitutional remedies (potassium iodid, mercury and salvarsan) should be administered. The iodid should be pushed to the point of toleration. Should alarming symptoms arise from edema, tracheotomy may become a necessity. It should be done low down so as to avoid the edematous tissues of the larynx. The tube should be left in place for some days, as its removal might be followed by an extension of the edema downward, thereby preventing its reinsertion. In the cicatricial type of stenosis the cutting dilator should be used and the O'Dwyer tube inserted. Daily dilatation with bougies may be practised instead of introducing the O'Dwyer tubes.

Tubercular Laryngeal Stenosis.—It is usually due to edema (Fig. 169). Cicatricial stenosis does not occur, as there is little tendency toward a reparative process. The treatment should be as for edema from other causes.

Lupus Laryngeal Stenosis.—*Pathology.*—The reparative process is prominent, hence *cicatricial bands* are apt to form. They sometimes almost completely close the glottis until the patient has barely enough space left through which to breathe or vocalize. Ordinarily respiration or vocalization are but little interfered with, as the bands are located above the cords.

Prognosis.—There is a tendency to spontaneous atrophy of the new-formed tissue, hence the prognosis is more favorable than in other types of stenosis. It should be remembered that there is some tendency toward the true tubercular type, which, if it occurs, renders the prognosis quite grave.

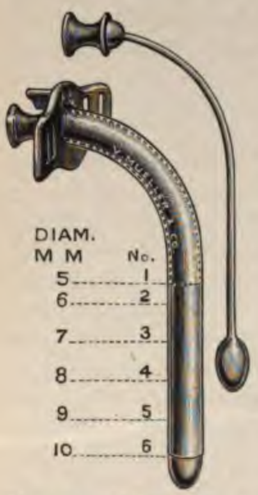

FIG. 168.—Jackson's cane-shaped trachea tubes.

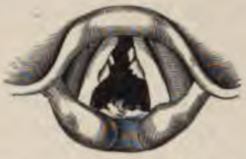

FIG. 169.—Tubercular infiltration of the interarytenoid space with tubercular papillomata of both vocal cords. Characteristic edematous infiltration of the aryepiglottic folds. (Cohen.)

Treatment. — Surgical measures should not be undertaken unless there is marked embarrassment to respiration. Dilatation is of little value unless preceded by cutting the web. So long as there is active inflammation, radical measures should not be instituted unless there is imminent danger of suffocation.

Traumatic Laryngeal Stenosis.—*Etiology.*—This condition may arise from intentional or accidental wounds to the throat; from foreign bodies; or caustic fluids within the larynx.

Symptoms.—The symptoms are those peculiar to interference with respiration and vocalization.

Prognosis.—The prognosis should always be guarded, as there is a tendency for the cicatricial bands to increase and suffocation thereby be produced. Edema may also develop and cause sudden alarming symptoms. The prognosis as to the voice should also be guarded, as the latter is apt to be seriously impaired.

Treatment.—The degree of the stenosis and the nature of its cause will determine the treatment. If due to granulation formations, these should be removed with laryngeal curettes. If due to edema, scarification should be done; and, if it fails to afford relief, tracheotomy must be performed. Cicatricial bands should be cut and O'Dwyer's tubes inserted daily.

INFLAMMATIONS OF THE LARYNX.

General Etiology. — *Mouth-breathing.* — Mouth-breathing may be due to enlargement of the pharyngeal and faucial tonsils; retropharyngeal abscess; deformities and neoplasms; also to congenital stenosis of the nasal fossæ; hypertrophy and swelling of the nasal mucous membrane; neoplasms; and foreign bodies.

Undue patency and consequent dryness of the nasal fossæ may be due to congenital arrest of development of the turbinal bodies; atrophic rhinitis; the surgical removal of the inferior turbinals; and collapse of erectile tissue of the nose.

The effects upon the larynx is much the same as that produced by mouth-breathing.

When the nasal and buccal secretions are *normal* the pharynx and larynx are protected to a great extent against disease.

Lymphoid Glands.—Infection of any portion of the lymphoid tissue of the pharynx, mouths of the Eustachian tubes, base of the tongue, the fauces, the larynx, the nasopharynx and nose frequently is the starting-point of a simple laryngitis.

Nasal Disease and Malformations.—Inflammations of the throat are frequently secondary to some nasal disease or malformation. Laryngeal inflammations are usually subacute extensions from the nares, and are associated with nasal stenosis. Microörganisms are usually the inciting cause.

Constitutional Conditions.—The rheumatic or gouty diathesis plays an important *role* in laryngeal diseases.

Improper Use of the Voice.—Various writers have recently called attention to the fact that the improper use of the voice among singers, public speakers, and criers accounts for most of the morbid conditions peculiar to their professions. Formerly it was held that the local morbid condition was the cause of the vocal disturbance, and that in order to effect a cure, rest of the voice and local, surgical, and medical interference were necessary. In accordance with the present view the rational treatment consists of the proper training of the vocal apparatus, thereby rendering it capable of performing the excessive amount of work put upon it.

The Influence of Improper Clothing.—The mode of life, habitation, and occupation should be taken into consideration in advising the patient as to the *kind* and *amount of clothing* he should wear. The amount of clothing should be modified to suit the immediate environment of the individual —*i. e.*, it should be so arranged that when in the house the clothing may be light in weight, conforming to the temperature of the room. While out of doors in severe weather outer coats should be used so as to protect the body in proportion to the change in environment.

Acute Simple Laryngitis.—**Etiology.**—*Age.*—The larynx is smaller in children, hence is more easily stenosed than it is in adults. While acute laryngitis is more frequent in adults than in children, it is not so dangerous. Enlargement of the postnasal lymphoid tissue predisposes to laryngeal inflammation. Plastic exudates are more common in children.

Climatic conditions, as cold, moist air, are exciting causes of inflammations here as elsewhere in the respiratory tract. The clothing should be so regulated as to protect without overheating the individual.

Improper or *overuse of the voice*, especially if there is lowered vitality from overwork, anxiety, grief, or sickness, may cause laryngitis.

Poor ventilation in shops, school-rooms, stores, and bedchambers predisposes to attacks of acute laryngitis. Fumes, gases, and dust may also irritate the larynx.

Disturbances of digestion and of the portal circulation which arise from the abuse of alcohol, tobacco, and food predispose to hyperemia and acute inflammation of the larynx.

Symptoms.—These will vary according to the location and amount of activity of the inflammatory process. If the aryteno-epiglottic folds and epiglottis are involved, as in laryngitis due to scalding drinks or liquors, *dysphagia* will be a prominent symptom. If the true cords and ventricular bands are prominently involved, there will be *dysphonia*. Should the interarytenoid space be the seat of inflammation, *cough* will be the prominent symptom. The cough may have a clear tone and the voice be almost free from hoarseness. In many of the so-called dry coughs attributed to chronic bronchitis, if the interarytenoid space (posterior commissure) were examined laryngoscopically, it would be found to be the seat of inflammatory thickening. *Hoarseness* is more or less prominent according to the amount of thickening of the vocal cords and impaired action of the laryngeal muscles. *Edema* and *hemorrhage* are more apt to occur in laryngitis of singers and public speakers than in persons of other callings. This is perhaps due in part to excessive use of the voice after the inflammatory process is established.

Age also modifies the symptoms, as in children there is a greater tendency to *spasmodic closure* of the glottis than in adults. This is especially true between the ages of two and five years. This may be accounted for in several ways, viz.: at that age there is a predisposition to the spasmodic action of the muscles from reflex irritations. The larynx is much smaller in the child than in the adult, hence the dried secretions which accumulate during sleep, especially if the child is a mouth-breather, may be sufficient to obstruct the glottic opening.

The *constitutional symptoms* are about the same as those in acute rhinitis and need not be detailed here.

Laryngeal Image.—Great variation will occur in the laryngeal image according to the particular location of the inflammation, the age of the patient, his occupation, and the stage of the disease.

Small areas or the whole membrane may be slightly thickened or red, the cords may remain a whitish yellow, being little or not at all thickened; or they may be quite red and swollen. At the onset the membrane is dry, but at the end of twenty-four hours a glairy mucous secretion is present. In some cases after a few days the secretion may assume a mucopurulent character. The mucous membrane, which at first presented an evenly red and swollen appearance, may in the later stages present patches of softened and desquamated epithelium. The submucous tissues may also be broken down beneath these spots. The ventricular bands may be so swollen that they overlap the true cords and interfere with phonation.

Prognosis.—Rest and the proper hygienic surroundings, if complied with at an early stage, will in most cases effect a spontaneous cure.

Treatment.—The patient should be positively instructed not to use his voice even in a whisper for a period of forty-eight hours. All communications should be made by signs or in writing. He should be confined within the house where the temperature is maintained at about 65° to 70° F., in which the atmosphere is kept heavily laden with moisture from boiling water. He should be taught that absolute relaxation of the vocal apparatus, and, indeed, the whole body, will aid materially in restoring his voice to perfect condition within a very short time.

In addition to the foregoing requirements, he may wear a wet compress, made of a small piece of folded linen, on his neck over the larynx. The compress should be held in place by a band of flannel a little wider than the compress and long enough to go twice about the neck. The compresses should be wet in cold water every four or five hours, squeezed slightly to avoid dripping, and applied as above described.

Four or five 4-grain doses of calomel should be given at intervals of two hours, after which free purgation should be produced by the administration of a full dose of Glauber or Epsom salts.

As violent coughing aggravates the inflammation, it should be allayed by the administration of small doses of codein. Should the secretions be very heavy and tenacious, the administration of $\frac{1}{100}$ grain of pilocarpine with very minute doses of antimony and ipecac is advisable.

If there is considerable tenderness over the larynx, the parts should be massaged freely with camphorated oil.

All patients should be instructed as to the necessity of protecting the feet from wet and cold by wearing heavy boots. A cold sponge-bath, followed by vigorous friction, should be indulged in every morning. In this way the activity of the skin will be maintained and the probability of future attacks be very much lessened.

One teaspoonful of the compound tincture of benzoin in one-half pint of boiling water may be used at intervals of from two to three hours as a steam inhalent to relieve cough and irritability. Bland sprays of oil are of some value. Kyle reports favorably on the use of dilute nitric acid in five- to ten-drop doses in water every half-hour for three doses, as a means of relief for those who are compelled to use their voices. The cold water or ice-bag applied over the neck in the early stages often relieves the distressing symptoms.

Surgical Treatment.—Should there be moderate edema of the epiglottis or mucosa of the larynx, they should be freely scarified with a laryngeal lancet. There is little danger of serious hemorrhage from this procedure, as the parts are filled with serous fluid rather than blood. Scarification should be followed by an application of mild astringents, as the glycerite of tannin. This form of astringent is especially valuable, as the glycerin promotes osmosis of serum from the edematous tissues, while it carries the tannin into the tissues. When there is imminent danger of suffocation, intubation or tracheotomy may be indicated to prevent a fatal issue.

Laryngitis Symptomatic of Constitutional Diseases.—**Erysipelas.**—In the early stages it resembles acute simple laryngitis. Later it extends to adjacent parts, sometimes even involving the face. Other exposed persons may become similarly affected. In the severer types the larynx may be affected by a phlyctenular or even a herpetic eruption. Rarely gangrenous areas develop.

Measles.—Usually there is a mild profuse redness with hoarseness and aphonia. A careful laryngoscopic examination will show the characteristic rash and swollen follicles. Should the voice be used in crying or talking, there may be frictional erosions. There is usually a cough, which in a few days is followed by dysphonia.

The *treatment* should consist of a massage of mercurial ointment over the larynx, wet compresses, and cathartics. In rare cases it may be necessary to resort to tracheotomy.

Scarlet Fever.—Laryngitis is less frequent in this disease, and is attended by no symptoms of especial importance.

Smallpox.—Small whitish prominences composed of necrotic areas of epithelium, under which there is a small amount of pus, form the chief objective symptom.

Typhoid Fever.—There is a deposit of specific bacilli, typhoid infiltration, and ulceration of the mucosa. The edge of the epiglottis is subject to erosions which may leave scars. They may in time produce laryngeal stenosis.

Influenza.—The whole respiratory tract is usually involved. The interarytenoid mucosa is especially prone to involvement, which accounts for the dry cough so common in this disease. It so nearly resembles whooping-cough that a mistaken diagnosis is often made. Laryngeal hemorrhage may occur. The true vocal cords may become eroded. Although the disease is quite active, abscess formation is rare.

Whooping-cough.—The anterior surface of the posterior wall of the pharynx and larynx are red and slightly swollen, hence the constant source of irritation which expresses itself in the form of a series of spasmodic coughs. The secretion is scanty and difficult to remove. Ecchymosis and hematoma are sometimes found in the submucous tissue. *Local treatment* is of little value.

Rheumatism.—There is violent pain in the arytenoid and other cartilages. The arytenoid cartilages are restricted in their movements, hence vocalization is more or less interfered with. There is swelling of the mucous membrane covering the cartilages. The presence of pain, the restricted use of the muscles of adduction and abduction, and pain and swelling over the arytenoids form a group of symptoms which should not be mistaken for true paralysis.

Typhus Fever.—Laryngeal inflammation sometimes occurs, and may be quite dangerous. The membrane is bright red or dark and velvety, and covered with a sticky mucopus. Deep ulceration may expose the cartilages.

Malaria.—Characterized by intermittent fever, hoarseness, redness, edema, and dyspnea. Should the glottis be markedly edematous, it should be scarified, and cold compresses applied to the neck. Astringent sprays should be thrown into the larynx. The usual antimalarial treatment should be faithfully carried out.

Laryngismus Stridulus.—**Etiology.**—This is a disease characterized by spasm of the glottis, accompanied by stridor. Disease of the larynx and trachea, whether it be inflammatory or non-inflammatory in character, predisposes to this condition. Many cases have been noted in which intestinal disorder seems to be the only assignable cause. Likewise the phenomena occurring during dentition in early childhood holds a close relation to the disease. Rickets, neuroses, faucial disease or tumor, nasopharyngeal adenoids, uterine disease, the uric-acid diathesis, elongated uvula, and necrosis of the vertebræ, may, through circulatory changes and reflex irritations, cause laryngismus stridulus. Pressure upon the pneumogastric or spinal accessory nerves may bring about very serious consequences.

Prognosis.—The prognosis may be quite grave, while in other cases the danger is remote. The treatment, however, should always be promptly and vigorously prosecuted, and in the interval between attacks the special causes be sought for and removed if possible. One attack is quite apt to be followed by others.

Treatment.—The first consideration should be the prompt relief from the spasmodic seizure. The child should have cold water splashed on his spine and hot water on the nape of the neck. The tongue should be seized with a napkin and pulled upon rhythmically one-fourth as many times as there are pulse beats, in order to excite the respiratory centres. If the spasm is not speedily relieved, the larynx should be examined for foreign bodies or other sources of irritation. The administration of from five to ten drops of ether will sometimes afford prompt relief. The mucous secretions should be removed with the finger or probang. The administration of oxygen under pressure is attended with very favorable results, as asphyxia is imminent. After a warm bath of ten minutes' duration, the child should be wrapped in a warm blanket and wiped dry with a towel introduced within the blanket. After the acute attack has subsided the patient should be carefully examined for postnasal adenoids or enlarged faucial and lingual tonsils. Indeed, the entire system should be subjected to a most careful examination by the best practitioners and specialists available. This is justified on account of the probable return of the disease and the serious nature of its causes.

Acute Epiglottitis.—Occurrence.—This condition is usually associated with inflammation of the adjacent tissues. The lingual tonsils are most often simultaneously inflamed.

Treatment.—The treatment is as given for acute laryngitis. Here, as in laryngitis, it is quite important that the nasal and postnasal spaces should be freed from obstruction or other diseased condition that interferes with their respiratory functions.

Membranous Laryngitis (*Membranous Croup and Exudative Laryngitis*). — **Definition.**—This is a form of membranous laryngitis not caused by the Klebs-Löffler bacillus.

Etiology.—The exciting causes may be microbic, chemical, or mechanical irritants. The disease may be sporadic or endemic. Previous inflammations of the larynx, and especially preëxisting nasal infections, favor the development of the process. Sanitary and hygienic conditions have more or less to do with the causation. Improper clothing and living in damp, cold rooms are especially fruitful of this type of inflammation.

There has been considerable controversy as to whether membranous laryngitis is not always a true diphtheria. The Klebs-Löffler bacillus is sometimes found in the false membrane, while the clinical history of the case presents none of the characteristic signs of diphtheria. On the other hand, the membrane is sometimes found to be entirely free from diphtheritic bacilli, while profound toxemia is present. We are hardly prepared to make a positive differentiation between the two diseases, and yet, for clinical purposes, it seems well to describe them separately.

Pathology.—At the onset the membrane is hyperemic and slightly swollen, as in an acute simple laryngitis. At the end of eighteen or twenty-four hours the exudate begins to coagulate upon the surface, the rapidly proliferating epithelium and leukocytes being entangled within its meshes. The pseudomembrane thus formed usually involves only the epithelial layer, the submucous tissue being unaffected. Occasionally, however, the submucous tissue breaks down, but this is probably due to interference with the circulation of the parts rather than to an extension of the plastic exudate beneath the epithelium. The exudate is usually above the glottis, and after it is well developed, is situated mostly on the true cords. As it travels downward from the fauces it attacks the epiglottis and ventricular bands before reaching the true cords.

Symptoms.—The febrile movement usually begins with a chilly sensation or a distinct chill, the temperature ranging from 100° to 104° F., the pulse being full and bounding. Anorexia and restlessness are early manifestations. It may thus be seen that the febrile movement is quite different from that of true diphtheria, although the temperature may range even higher. The patient complains of the throat feeling stiff and sore, especially upon deglutition. The pains radiate from the larynx toward the ears. The temperature does not change materially during the first twenty-four to forty-eight hours, but as the larynx becomes covered with the membrane, it rises one or two degrees. The voice is quite characteristic, having a metallic ring upon coughing or attempted vocalization, and may become completely aphonic. This symptom will vary with the amount and location of the false membrane and with the degree of muscular impairment. Dyspnea is present in proportion to the amount of laryngeal stenosis, and is both inspiratory and expiratory. It is attended by the usual signs, as cyanosis, supraclavicular depression, anxious expression of countenance, extreme restlessness, and dilated nostrils. It is progressive until death ensues or the membrane is thrown off. The membrane may reform, hence extreme care should be observed until convalescence is well established. The natural course of the disease is from three to six days, the larynx being invaded about the second or fourth day, when the suffocative symptoms appear. The presence of albumin in the urine is not significant of true diphtheria, as it may be found in simple follicular inflammation of the tonsils. The same may be said of paralysis of the palatine muscles.

Diagnosis.—The disease with which it is usually confounded is diphtheria; and as the presence or absence of the Klebs-Löffler bacillus does not afford a positive means of diagnosis, it is well to give the patient the benefit of the doubt and treat him as for diphtheria. Bacteriologic examination of the secretion and fragments of the membrane should be made early in the disease, but failure to find them should not be taken as sufficient data upon which to discard diphtheritic treatment.

Other diseases which it may resemble in some respects are spasmodic laryngitis, laryngismus stridulus, and retropharyngeal abscess.

Treatment.—In the early stages of the disease, before the formation of croupous membrane, *cold* applied to the outside of the neck will sometimes abort the disease. It may be applied by means of a long, narrow ice-bag partially filled with cracked ice, or by means of a Leiter coil made of lead tubing so bent as to conform to the shape of the neck. If the ice-bag is used, it should be wrapped with a piece of thin muslin, and should be refilled every hour. If the Leiter coil is used, it should be connected by means of a siphon tube with a basin of ice-water placed on a higher level than that of the patient. In this way the neck is subjected to the action of the continuous current of cold water. Heat may be used by either of the above methods, but is ordinarily less efficacious than cold.

The *atmosphere of the room* should be kept at a temperature of about 70° F. and surcharged with lime vapor. A good method of administering lime vapor is to slake a pound or two of lime in a vessel of water by the side of the bed, a temporary tent, made of a sheet, being so held as to cover the child and lime vessel. The child should be thus compelled to inhale the vapor of the lime for ten minutes, at intervals of one to three hours. If lime is not available, the air of the room should be surcharged with steam from boiling water to which have been added carbonat of sodium and spirit of turpentine. Steam may also be administered directly to the child by means of an atomizer placed on a stand by the side of the bed. These and other remedies, as the compound tincture of benzoin, creosote, oil of eucalyptus, and the benzoate of sodium, may be thus administered for the relief of cough and the thick, tenacious secretion.

Internal Medication.—In this, as in exudative inflammation of the tonsils, the mixture containing two parts of the tincture of chlorid of iron and eight parts of glycerin should be given in half-dram doses every two hours to a child five years old. Calomel should be administered in broken doses until free catharsis is established.

Should laryngeal stenosis become so marked as to produce profound dyspnea and cyanosis, either *intubation* or *tracheotomy* should be immediately performed.

Perichondritis of the Laryngeal Cartilages.—Definition.—This is an inflammation of the laryngeal cartilages which may be characterized by ossification, hypertrophy, fibrous thickening, ulceration, or abscess.

Etiology.—It is usually due to syphilis, tuberculosis, acute infections, rheumatism, and traumatism. Children and young adults are rarely affected, it being most common after middle life. Men are more often affected than women.

Pathology.—In the early stages there are the usual inflammatory changes, which finally terminate in ossification, hypertrophy, hyperplasia, ulceration, or abscess. Sometimes the opening of the ulcer into the cavity of the larynx is quite small, while the tissues beneath are undermined in all directions for quite a distance. The amount of damage done cannot always be estimated by the apparent size of the ulcerous opening. In all cases the muscular movements of the larynx are interfered with, both on account of the articular changes and the changes within the muscles themselves.

Symptoms.—The acute stage is ushered in with a febrile movement as in acute simple laryngitis. There are chilly sensations, malaise, headache, loss of appetite, pains in the bones, and a temperature of 100° to 101° F. The vocal functions are impaired according to the degree and location of the inflammation.

In the syphilitic type there are marked tenderness and pain upon pressure. If the cricoid cartilage is involved, the perichondrium may be distended inward, closing the lumen of the larynx, thereby producing dyspnea. When the posterior part of the cartilaginous box is inflamed, dysphagia becomes a prominent symptom on account of the close proximity to the esophagus. Important laryngeal muscles may become infiltrated, and the voice impaired or lost. Should one of the arytenoid cartilages be the seat of inflammation, laryngoscopic examination would show a one-sided tumefaction. This is a common location in tubercular perichondritis, the voice becoming hoarse and low in pitch. When the thyroid is involved, the voice becomes hoarse or aphonic, and the respiration dyspneic. As the epiglottis is a fibrocartilage, it is rarely the seat of perichondritis. When the perichondritis assumes the ulcerative form, there is a tendency to the formation of a fistulous opening outward through the tissues of the neck, or internally into the larynx or adjacent tissues. The necrotic cartilage is exfoliated through the fistula thus formed.

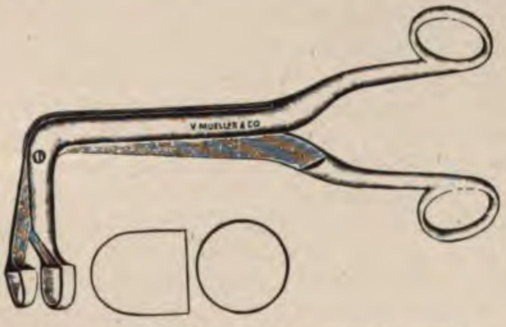

FIG. 170.—Moritz-Schmidt's epiglottis punch.

Prognosis.—The course is usually a chronic one, and may lead to death by suffocation. In the acute type the attack is sudden and life may be threatened within a few hours.

The prognosis is more or less affected by the constitutional dyscrasia causing the disease.

Treatment.—The treatment should be varied according to the etiology of the particular case. In a general way it may be said that antiphlogistic measures should be instituted, as cold applications, leeching, wet cupping, etc. If there is a sluggish portal circulation, calomel should be administered until catharsis is produced, when a saline draught should be given. In the ulcerative type there should be an attempt to stimulate nutrition and assimilation, hoping thereby to arouse the reparative energy of the blood and cellular tissues.

Where there are ulceration and exfoliation of the cartilage, surgical interference may be demanded. The exfoliated cartilage should be removed at once, either through the tissues of the neck or through the mouth. Tubercular ulcers should be curetted and rubbed with a 2 to 10 per cent. formalin solution or trichloracetic acid. If marked dyspnea arise, intubation or tracheotomy should be performed.

Chronic Simple Laryngitis.—Definition.—This is a chronic superficial inflammation of the mucosa, including the glandular, vascular and connective-tissue elements, characterized by hoarseness or aphonia.

Etiology.—This disease is rarely primary, but is nearly always secondary to some form of disease of the nose, nasopharynx or faucial tonsils. It is very important that this part of the etiology be well understood, as the principal part of the treatment is based upon it. Any condition of the nose or throat which interferes with the respiratory functions will bring about a lowered power of resistance of the laryngeal mucous membrane, thereby making it a favorable location of catarrhal inflammation. The respiratory functions of the nose being lost or impaired, the inspired air passes to the larynx insufficiently warmed, humidified, and filtered, and irritates the larynx and lower air tract by overtaxing its glandular and circulatory apparatus. The conditions of the nose which impair its functions may be obstructive, as hypertrophic rhinitis, cystic or polypoid degeneration of the middle turbinal, neoplasms, rhinoliths, foreign bodies, and deflection of the septum. Diseased conditions which are not obstructive, as simple dry catarrhal rhinitis, atrophic rhinitis, or ozena, may also impair or destroy the respiratory functions of the nose. Hence it is important that intranasal conditions should be carefully examined for when trying to determine the causes of chronic laryngitis. In suppuration of the posterior ethmoidal cells and sphenoidal sinuses the pus discharges over the middle turbinal into the nasopharynx, and drops down upon the laryngeal mucosa, where it acts as an irritant. It may thus be the chief cause of laryngitis. Other causes which are more or less responsible for chronic laryngitis are improper clothing, unhygienic environment, unfavorable climatic conditions, the rheumatic diathesis, the excessive use of alcohol and tobacco, repeated attacks of acute laryngitis, and overuse or faulty use of the voice.

Esophagitis and gastritis have been known to cause chronic laryngitis. Sluggish circulation in the portal system causes a hyperemia of the upper respiratory tract, thereby favoring the inflammatory process.

Symptoms.—The most characteristic symptom is the appearance of hoarseness in the morning after very moderate use of the voice. During the day the voice may regain its clearness, but soon relapses into the hoarse condition. Toward evening the hoarseness is especially marked and annoying. Public speaking is attended or followed by hoarseness or complete loss of voice. The amount of hoarseness is somewhat dependent upon changes in the weather. The patient complains of a slight sore feeling in the upper part of the laryngeal box, and will sometimes suggest that it is rheumatism. There is a sensation of "a web-like formation over the vocal cords, which, if removed, would render the voice quite clear." The patient unconsciously attempts to remove the imaginary web by clearing the throat. It should be explained to him that by so doing he gets no relief, but, on the contrary, increases the irritation and perpetuates the laryngitis. Those who have impairment of the nasal respiratory functions may be markedly neurotic; hence in chronic laryngitis the nervous element may be of considerable importance, and should receive appropriate treatment. After slight use of the voice the patient has a feeling of muscle-tiredness.

Upon laryngoscopic examination the mucosa appears thickened, sometimes in its whole surface, and at other times in limited areas only.

The foregoing symptoms vary somewhat with the location and extent of thickening. If the interarytenoid space is thickened, a dry cough accompanies the hoarseness.

Diagnosis.—Some of the symptoms of this disease are common to tubercular, edematous, paralytic, syphilitic, and malignant disease of the larynx.

Edema of the larynx presents a membrane of much the same color, but more swollen, with a history of sudden onset and dyspnea.

Paralysis of the vocal cords is attended by inability or impaired ability to move the arytenoid cartilages.

Tubercular laryngitis in almost every case is secondary to a pulmonary tuberculosis.

In *syphilitic laryngitis* the history and presence of the diffused edema of the larynx, the Wassermann test and a favorable response to antisyphilitic treatment, render the differential diagnosis comparatively easy.

In *malignant disease* of the larynx the diagnosis in the early stage is rendered somewhat difficult on account of the late appearance of glandular enlargement, edema, and ulceration. A careful examination of the larynx will, however, reveal an enlargement of the interior of the larynx, usually on one of the vocal cords which in case of a superficial growth can be confirmed by a microscopic examination.

Prognosis.—The prognosis of chronic laryngitis, so far as the recovery of the voice is concerned, should be guarded, as a favorable termination depends very much upon conditions over which the physician has no control. If the cords have not become markedly thickened, and the patient will submit to the surgical treatment needed to restore nasal respiration, and will give his vocal apparatus the needed rest, there is a very good chance for the restoration of his voice. Although the hoarseness seems to be due to a simple laryngitis, the prognosis should be guarded if there is a family history of tuberculosis. In many cases the voice remains permanently hoarse in spite of all forms of treatment.

Treatment.—After giving attention to the clothing, diet, bowels, vocation, and special dyscrasiæ, the nasopharyngeal abnormalities should receive appropriate treatment. *Local palliative treatment* in the form of astringent inhalants or atomized fluids, containing benzoin, eucalyptus, and camphor, should be used to allay cough, sense of irritation, and cobweb sensation in the larynx. *Local painting*, with 10- to 20-grain solutions of the mineral astringents, as the nitrate of silver, sulphate of copper, and chlorid of zinc, exert a favorable influence upon the progress of the disease. The tenacious secretions may be somewhat thinned by the administration of an expectorant. The use, or at least, the abuse, of alcohol and tobacco should be interdicted.

The tonsils should be carefully examined as a possible etiologic factor.

To *recapitulate*, the larynx should be given a rest, the nasopharyngeal disease removed, distressing symptoms palliated by the proper use of local remedies, nervous symptoms and constitutional disorders corrected, and, most important of all, obstructive and diseased conditions of the nose and throat should be radically corrected.

Follicular Laryngitis. — **Definition.** — This is a type of chronic laryngitis in which there is hypertrophy of the acinotubular glands and the lymphoid tissue of the laryngeal mucosa.

Occurrence.—The condition is comparatively rare, and it is quite similar to follicular pharyngitis. It is most common in those who make a professional use of the voice, and it has often been called "preacher's laryngitis."

Treatment. — The administration of small doses of the syrup of the iodid of iron stimulates the mucous glands, thus relieving the congestion and thinning the secretions. The patient should be taught the proper use of the vocal apparatus and cautioned against its abuse.

Atrophic Laryngitis.—**Synonym.**—Laryngitis sicca.

Etiology.—This condition is one which is usually secondary to some disease of the nose or pharynx. Atrophic rhinitis is the most common cause. Deflection of the septum, tumors, hypertrophic turbinates, etc., may also cause it. Dry laryngitis is more common in females. A dry pharyngitis may also be associated with laryngitis sicca.

Symptoms.—During waking hours the movements of the vocal apparatus keep the crusts from accumulating, hence the symptoms are comparatively slight. On waking in the morning there is more or less loss of voice on account of the presence of the crusts and inspissated mucus. If the crusts are large, there may be pronounced dyspnea. Their presence causes a sense of irritation and cough. Upon inspection with the laryngoscopic mirror greenish-yellow crusts may be seen below the cords, although they are occasionally above the cords. The breath is fetid upon *oral* exhalation. It is necessary to make a distinction between oral and nasal exhalation, as the nose imparts its fetid odor in nasal exhalation. Sometimes there are erosions of the mucosa under the crusts; hence when the crusts are expelled by coughing, there may be slight hemorrhage.

Prognosis.—The prognosis depends largely upon the curability of the nasal and pharyngeal diseases causing it. If the atrophic rhinitis is in the early stages, there is a fair chance of effecting a cure; if, however, it is well advanced, the prognosis is bad. If it is due to deflection, spurs, ridges, or intranasal tumors, the prognosis is good after the removal of such, provided that the glandular elements are not too badly diseased. Pharyngitis sicca is rather intractable to treatment; therefore a dry laryngitis secondary to it is also intractable.

Treatment.—It is of prime importance that the intranasal disease causing it should be remedied. The atrophic rhinitis, sinusitis, septal abnormalities, tumors, etc., should be corrected if it is possible so to do. Any diseased condition of pharynx and nasopharynx should also receive appropriate treatment.

The *local treatment* of the larynx should consist of detergent sprays (Dobell's or Seiler's solution) followed with mentholated oil at intervals of three or four hours. This may be done by the patient. The physician should make applications of astringent and slightly stimulating lotions to the larynx. For this purpose weak solutions of silver nitrate, sulphate of zinc, compound tincture of benzoin, etc., may be used. The treatment is usually a protracted one.

The internal administration of the iodids will stimulate the mucous glands and render the disease more tolerable.

A *change of residence* to a climate with a uniformly humid atmosphere affords great comfort.

Hypertrophic Laryngitis.—**Etiology.**—This is a comparatively rare disease, and is the result of prolonged over-nutrition of the parts from persistent hyperemia.

Pathology.—It has been described by some writers as a scleroma, but a true scleroma is characterized by an excess of fibrous tissue, attended by ulceration and contraction; while in true hypertrophy all the elements of the mucous membrane are increased and there is no tendency to contraction.

Symptoms.—The movements of the vocal cords are interfered with according to the location and degree of hypertrophy. If it is over the arytenoid cartilages, vocalization is impaired, while respiration may remain perfectly free. Should, however, the hypertrophy involve the ventricular bands to such a degree that they project over the vocal cords, not only will vocalization be impaired, but respiration as well. The hypertrophic process, however, is located most often in the posterior commissure, where it may be seen as slightly nodular or granular eminences, projecting upward and forward into the chink of the glottis. When in this location, the motility of the cords is somewhat impaired and cough is a more or less prominent symptom. Esophagitis sometimes causes this condition by an extension of the inflammatory process, and should therefore receive appropriate treatment.

Treatment.—The treatment of hypertrophic laryngitis is of comparatively little value. If the hypertrophy is excessive, it may be removed by means of laryngeal cutting forceps, chromic-acid applications, or the actual cautery. Careful examination should be made for the causes of the condition and measures addressed to their relief, hoping thereby to arrest the progress.

Edematous Laryngitis (*Phlegmonous Laryngitis; Submucous Laryngitis*).—**Definition.**—This is a form of laryngitis attended by a serous infiltration of the submucosa of the larynx and epiglottis. Occasionally the mucosa of the trachea immediately beneath the cords is also involved.

The *predisposing causes* are (*a*) male sex; (*b*) early adult life; (*c*) general debility; and (*d*) great fatigue.

The *exciting causes* are "colds;" a mild acute, simple laryngitis; exanthematous fevers; irritating vapors or chemicals; steam inhalations; foreign bodies; trauma; syphilitic or tubercular infections; retropharyngeal abscess, etc. It is sometimes secondary to prolonged intralaryngeal operations or examinations.

While it is usually found in young adults, it occasionally occurs in infants and elderly people.

Pathology.—The inflammatory products of acute laryngitis, plus vascular turgescence and serous infiltration of the loose submucous tissue, are present. The parts most affected, therefore, are the aryepiglottic folds, ventricular bands, and the posterior surface of the epiglottis. The cords are usually free from infiltration, but are red from vascular congestion. The swelling is usually symmetrical. During the late stage of the disease there may be pus formation in the submucous tissue.

Symptoms.—It is generally ushered in by chilly sensations or a distinct chill. A febrile movement of 100° to 103° F. is established. Dyspnea immediately follows the onset of the fever, and it may progress gradually or develop suddenly until extreme distress and danger to life are imminent. The voice is rough and heavy, while respiration is difficult. In the beginning the obstruction to the breathing is inspiratory. Later the expiratory effort is also obstructed. Extreme anxiety then becomes a prominent symptom. The dyspneic symptoms last for about twelve to seventy-two hours. Cough is incomplete and ineffective. Deglutition may be difficult and distressing. The appearance of the edematous and inflamed laryngeal mucosa depends somewhat upon the exciting cause of the disease. The peculiar eruption of the exanthem, the character and nature of the traumatism, will modify the appearance of the infiltrated mucosa. In a general way the infiltrated and inflamed mucosa presents a globular semitransparent appearance with a bright red circumference. The capillaries of the epiglottis are injected and the vocal cords are red and somewhat thickened. When the membrane below the glottis (infraglottic) is edematous, it is still more red than the cords.

Prognosis and Course.—The disease usually develops rapidly, and may lead to a fatal issue within a few hours. The dyspnea generally, however, lasts for three or four days. The danger is almost always from asphyxia. Occasionally an abscess or gangrenous process leads to a fatal termination. Edema of the infraglottic mucosa renders the prognosis very grave.

Treatment.—The most important indication is to overcome the dyspnea and thereby avoid suffocation. The most efficient method of accomplishing this is by means of a laryngeal lancet. The scarification should be repeated three or four times a day to unload the serum from the distended membrane. Some benefit may be derived from the use of steam inhalations and pellets of ice in the mouth. Leeches applied over the cricothyroid membrane, and the ice-bag or the Leiter coil applied continuously are valuable adjuncts to the treatment. If pus is present, it should be evacuated. If the dyspnea is progressive and the patient becomes cyanotic, tracheotomy should be done. It should be done low down on account of the possibility of there being infraglottic edema. After the tracheotomy-tube is introduced, it should be allowed to remain as long as there seems to be edema, as the parts about the wound might become edematous in its absence and render its reintroduction difficult or impossible.

Simple Edema of the Larynx. — **Synonym.** — Edema Glottidis.

Definition.—Simple edema of the larynx is unattended by marked inflammation of the larynx. It is a serous infiltration of the loose submucous tissue of the larynx.

Etiology.—It is usually due to the same causes that produce edema in other parts of the body. Some form of kidney disease is the most common cause. Cardiac weakness or insufficiency, vasomotor disturbance of the capillary circulation, hepatic disease, etc., may also be the cause.

Symptoms.—The onset is sudden, and the signs are those of inspiratory obstruction. This gradually increases with intervals of comparative comfort for two to four days, when a fatal issue may occur if the remedies prove ineffectual. The dyspnea is accompanied by stridulous inspiration. After the first six to twelve hours the dyspnea becomes quite marked, hoarseness and aphonia develop, the patient becomes restless and assumes an anxious expression of countenance. There is a sense of fulness and tension in the throat, with slight pain upon swallowing. The *laryngoscopic image* reveals the mucous membrane in the region of the epiglottis, the aryepiglottic folds and sometimes the subglottic region to be swollen and of a pale gray color, in marked contrast to the tumefaction in edematous laryngitis in which it is red.

Prognosis.—The prognosis depends somewhat upon the cause of the local disease. If it is due to local conditions of the pharynx and upper air tract, the prognosis under treatment is very good. If it is due to syphilis or tuberculosis, the prognosis as regards immediate danger is fair. This is especially true of syphilitic edema, as it is more amenable to constitutional treatment. The "angioneurotic edema" (urticaria of the larynx) is of transient duration and generally ends in recovery. If, however, the laryngeal edema is due to nephritis, or cirrhosis of the liver, the ultimate prognosis is bad.

Treatment.—If two facts are borne in mind, the indications for treatment become quite apparent. The two facts are (*a*) the local condition is one of serous infiltration in the loose areolar tissue of the larynx and epiglottis; (*b*) the edema is usually due to some general disease which may produce edema in other parts of the body.

The first fact suggests the propriety of puncturing the edematous masses in order to relieve the intense tumefaction which causes respiratory obstruction. The laryngeal lancet may be used for this purpose. Repeat the scarifications every three to five hours.

The second fact suggests the propriety of treating the general disease causing the local laryngeal edema. Intubation, catheterization, or tracheotomy should be performed if cyanosis occurs. The syphilitic and tubercular types rarely advance so far as to require surgical interference.

Pachyderma of the Larynx.—Definition and Pathology.—This is a form of *chronic laryngitis* characterized by the formation of pavement-epithelium on the free borders of the cords and in the interarytenoid space. The thickening of the epithelium is progressive; hence the peculiar "tumor" formations are developed. The subepithelial connective tissue is also thickened. The growths are usually on the cords along the posterior third—*i. e.*, that portion of the cords corresponding to the arytenoid cartilages. The next most common location is in the interarytenoid space posteriorly. They are also occasionally found at the junction of the anterior and middle thirds of the cords. When located in this region they are known as *singer's nodules*.

Symptoms.—The symptoms are those of chronic laryngitis associated with those of dry laryngitis. When the thickenings are on the posterior third of the cords, one of them fits into a corresponding facet on the opposite tumor. On this account they do not interfere with the voice as much as would be expected from so large a lesion.

Prognosis.—These thickenings do not affect longevity. If they are located on the posterior portions of the cords, the voice will probably remain somewhat impaired, especially for singing. If they are on the anterior two-thirds of the cords, they can be made to disappear so as to leave the voice unimpaired.

Treatment.—The inflammatory condition of the nose and pharynx should be attended to. Potassium iodid has been recommended. *Locally*, a spray of normal salt solution, or of a 2 or 3 per cent. solution of acetic acid, is of value. In some cases the acetic-acid solution will be too irritating. The perchlorid of iron, 2 drams to the ounce, or nitrate of silver, 1 dram to the ounce, may be of some value in reducing the growths and relieving the symptoms. If, however, the "tumors" are large, they should be removed by surgical interference. As singer's nodules are due to the improper use of the vocal apparatus, they may be permanently cured by vocal exercises suited to the case.

Singer's Nodules on the Vocal Cords (*Chorditis nodosa*).—Reference has just been made to this condition in the preceding section. These nodules occur as the result of the improper use of the voice, and especially from exercises in which the cords are approximated, as in the production of the syllable ah. This exercise is known as the "stroke of the glottis." Its proper use gives brilliancy to the voice, while its improper use impairs it in the middle register. The cords are close together and nearly parallel when singing in this register, hence the nodules interfere with their proper vibration. Singing exercises which avoid the close approximation of the anterior two-thirds of the cords, as the use of the vowel O or broad "a" *preceded by* the consonant "m," as "ma," will occasionally, after two to four weeks, cause the nodules to diminish in size or to disappear.

Abscess of the Larynx.—**Treatment.**—It should be opened with a laryngeal lancet, the patient immediately throwing the head forward and downward to prevent the pus entering the trachea. If this precaution is not observed, suffocation may ensue. After a few moments of embarrassed respiration the desired relief follows.

Tuberculosis of the Larynx.—**Etiology.**—The *predisposing causes* are the same as those of tuberculosis in other organs of the body.

It is possible to have a primary tuberculosis of the larynx, although rarely seen. The vast majority of cases are secondary to a pulmonary involvement. Some few cases may be secondary to a lymphatic involvement, especially of the tonsils and cervical glands.

The disease is more common in men than in women and occurs most frequently between the ages of twenty and forty years.

Local disease of the larynx probably favors its manifestation, although this view is disputed by some writers. Tubercular laryngitis accompanies from 15 to 35 per cent. of the cases of advanced pulmonary tuberculosis. It rarely coexists with tuberculosis elsewhere in the body.

Pathology.—Four forms of the disease are usually met with. They are: infiltration, ulceration, tuberculoma and miliary tubercle. The infiltration most often takes place in the interarytenoid space, spreading gradually to the arytenoid cartilages and superimposed mucosa, ventricular bands, and epiglottis. It may be limited to one side and later spread to the opposite side. It is usually bilateral.

The ulcer follows the infiltration stage and is primarily due to a breaking down of one or more of the subepithelial tubercles (Lockard). The ulcer may enlarge by a continuation of the degenerative process or by the coalescence with others. Granulations cover the base and edges of the ulcer. Occasionally these granulations become exuberant. Flat superficial ulcers sometimes result from local irritations as from the action of the irritant sputum on the mucous membrane.

The tuberculoma is a tuberculous granuloma resembling a true tumor. It is rarely seen, and when present is usually on the posterior wall, the anterior commissure, or the ventricle of Morgagni. Microscopically, the nodules or tubercles composing the tuberculoma consist of numerous small round cells, giant cells and bacilli. Frequently a caseous degenerative process is present.

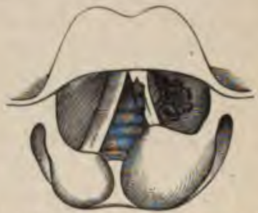

Fig. 171.—A tubercular ulcer on the left ventricular band and left vocal cord. Pear-shaped edematous swelling of aryepiglottic folds, more intense on the side of the ulceration. (Cohen.)

The miliary tubercle is rarely seen, but it is undoubtedly present in a few cases. It may be recognized macroscopically by the numerous minute, grayish-yellow or white spots clustered over the infiltrated area. These tubercles soon break down and ulcerate.

Symptoms.—*Functional.*—The loss of the voice is an early and frequent symptom. It sometimes changes suddenly from a gruff hoarseness to a high falsetto and thence into a toneless whisper. Dyspnea may occur in advanced cases on account of the great amount of infiltration. There may be cough and pain followed by extreme exhaustion. Tubercle bacilli are usually found in the sputum, but their absence does not necessarily prove the case to be non-tubercular.

Deglutition is impaired and attended by pain in advanced cases. The larynx is sometimes tender to the touch. Laryngeal hemorrhage is rare. The edema if present is moderate, and in nowise forms a serious complication. The pain frequently radiates to the ear.

Physical.—The mucosa in the early stages is frequently described as having a dull grayish-yellow or ashen color. This is probably a local manifestation of a general anemia. The cords may be red and the vessels of the mucosa injected. The arytenoid cartilages and the mucosa covering them become infiltrated and club-shaped. When in this condition they are usually referred to as pyriform swellings. After a time they break down or ulcerate, and are covered with a thick, tenacious, ropy secretion slightly mixed with pus. The arytenoid cartilates may become ankylosed and vocalization still further interfered with.

Prognosis.—The course of the disease is slow; but inasmuch as the disease is almost always associated with pulmonary phthisis the prognosis is grave, though not necessarily fatal.

Treatment.—This should be as for pulmonary tuberculosis with the addition of local measures. The treatment depends upon the stage and location of the process. If it is in the pre-ulcerative stage, simple alkaline washes or sprays should be used locally, so as to remove the secretion and stimulate regenerative processes. Liquor formaldehydi, used in strength of from 3 to 10 per cent., thoroughly rubbed in the infiltrated area is of value.

Most cases are not seen until the *stage of ulceration* is well developed; hence this condition is usually the one presented for treatment. An effort should be made to excite mild reactionary regeneration of the ulcerated surface. This can be done by first cleansing the parts with alkaline washes. The parts should then be dried with a cotton-wound applicator and the ulcers painted with a saturated solution of trichloracetic acid. The application of the acid should be made at intervals of from five to seven days. The ulcers are sometimes very deep and extensive. In such cases the ulcers should be curetted deeply with a laryngeal curette, care being taken to remove all of the tuberculous tissue. The limiting membrane which surrounds the diseased area will be broken, and if all tubercular tissue is not removed it may extend to the adjacent tissues. Mere curettement is worse than useless, but the thorough removal of the diseased tissue may be of the greatest value. The application of acids after curettement is of value chiefly as a destructive agent to the remaining fragments of tubercular tissue, and it also excites phagocytosis which raises the vital resistance of the parts.

The galvanocautery is a valuable weapon for local treatment. St. Clair Thompson reports the galvanocautery indicated in 20 to 22 per cent. of his 178 cases and that it completed the cure in 41 to 61 per cent. of the cases in which it was employed.

Pain is a most distressing symptom in many cases, especially upon swallowing. In such cases liquid or semisolid foods should be given and such anodyne remedies as afford the greatest amount of relief. For this purpose cocain sprays or powders containing 10 per cent. of cocain should be applied to the laryngeal mucosa. In very aggravated cases morphin may be used locally and by hypodermic injection.

In dysphagia from ulceration of the epiglottis, amputation of this structure is often followed by the greatest relief.

Injection of the recurrent laryngeal nerve with warm 80 per cent. alcohol has been done to relieve the pain. The patient is placed in the prone position, the head slightly extended and the chin turned away from the point of injection. The nerve is situated just above the upper edge of the thyroid cartilage and about 3 cm. from the incisure thyroideæ.

Locally, oily sprays containing 10 per cent. of menthol add to the comfort of the patient.

Pemphigus of the Larynx.—This is a rare condition, characterized by an herpetic eruption of the larynx, usually limited to the ventricular bands.

There is a slight constitutional disturbance, with local sensation of heat. The condition is often attended by gastro-intestinal disorder, the regulation of which should form a prominent part of the treatment. The mouth and pharynx should be frequently washed with an antiseptic gargle.

Syphilis of the Larynx.—As the *initial lesion* is rarely, if ever, found in the larynx, consideration will be limited to the secondary and tertiary stages.

Pathology.—*Secondary* syphilitic manifestations are *erythema*, the *mucous patch*, the *superficial ulcer*, and *condylomata*.

Erythema of the laryngeal mucosa is not usually coincident with the skin eruption, but follows it after a period varying from a few weeks to six months. The administration of the iodid of potassium causes its rapid disappearance.

The superficial ulcer is irregularly oval, extending its borders slowly. One or more ulcers may form at the same time, or may appear after the disappearance of the preceding ulcer. On account of this peculiarity it has sometimes been called "recurrent ulcerative laryngitis." After the subsidence of the ulcerative stage a superficial stellate scar is formed. The superficial ulcer occurs most often between the second and seventh years after infection, and yields readily to the classical treatment.

The mucous patch is rare, and according to some authors never appears upon the larynx. Those who have described it say that it always appears above the glottis.

Condylomata appear as yellowish pimples with elevated bases. They soon break down and discharge their contents, causing little or no discomfort or functional disturbance.

The changes characteristic of the *tertiary* stage rarely take place before the fifth year, but usually between the tenth and twenty-fifth years, although they may occur at a much later period.

The *tertiary* phenomena are *gumma*, *deep ulcer*, and *cicatricial stellate scars*.

The *gumma* is a small round-celled infiltration which may be either diffused or circumscribed.

The *diffused infiltration* involves the submucosa, muscles, and perichondrium; hence the movements of the larynx are interfered with. The infiltration occurs slowly, the membrane being red and sometimes edematous. The edema is a serous infiltration of the submucous tissue, and may obscure the true nature of the disease. After a time the diffused infiltration may undergo degenerative changes and become irregular superficial ulcers.

The infiltration may be *circumscribed* and stand out from the surface of the mucosa as a clearly defined tumor, or true gumma. The circumscribed gumma persists but a short time, as it usually undergoes rapid degeneration. The breaking-down process begins in the centre of the new formation in the form of fatty degeneration, which rapidly spreads to the periphery of the growth, thus merging into the ulcerative stage.

The *deep ulceration* which results has clear-cut, punched-out edges with perpendicular walls and a greenish-gray bottom. The ulcers throw off a foul-smelling semifluid discharge. One portion of the membrane may be in the ulcerative stage, while other portions are still in the stage of infiltration or gummatous swelling. The ulcers often form upon the upper surface of the epiglottis, and on the true cords, as these are the most exposed portions of the larynx.

Postulcerative or cicatricial scars form in the parts previously destroyed in the ulcerative process. The cicatricial bands usually radiate from the centre of the old ulcer, thus forming the peculiar *stellate scars* which are so characteristic of syphilis of the mucous membrane. The radiating bands show a marked tendency to contraction, and in consequence the tissue about them becomes much distorted. Stenosis of the larynx may occur, and suffocative symptoms intervene, which may require speedy tracheotomy to prevent a fatal termination. Mixed infection may remain long after the syphilitic inflammation has entirely subsided, and may require special treatment for its removal.

Symptoms.—The symptoms of the *period of infiltration* extend over several weeks or months, as it progresses very slowly. There are slight discomfort and pain upon swallowing, and a tickling or scratching sensation in the throat. The voice is more or less hoarse or aphonic according to the degree and location of the infiltration. If it is of the circumscribed sort, the motility of the vocal cords will be impaired. If the true cords are involved, there will be partial or complete aphonia. Strangulation upon attempts to swallow fluids may occur in extensive infiltration of the epiglottis.

In the *stage of ulceration* the pains become more marked, and are stabbing and fiery in character, especially after taxing the voice or upon awakening in the morning. The movements of the epiglottis may be circumscribed on account of the infiltration of its substance, and of the muscles controlling it; hence dysphagia may become a distressing symptom.

The symptoms of the *cicatricial stage* are those due to interference with vocalization, respiration, and deglutition.

Diagnosis.—It may be confounded with tuberculosis and malignant diseases of the larynx. The general symptoms and history of the case, when considered in connection with the local phenomena and the Wassermann and Noguchi tests, afford a ready means of diagnosis.

In *tuberculosis* the presence of tubercle bacilli in the sputum, the pulmonary signs, and the local laryngeal appearance form a symptom-complex, which should not be mistaken for syphilis.

Malignant disease of the larynx is more apt to be characterized by paralysis of one or both vocal cords, and by a cachectic appearance, which, in the absence of a syphilitic history, renders the diagnosis comparatively easy.

Treatment.—For the treatment of laryngeal syphilis main reliance should be placed upon mercury, especially in the early stages, and potassium iodid in the gummatous stages, and upon salvarsan or one of its equivalents.

In the early stages the local application of calomel or iodol are very agreeable. Mild alkaline antiseptic sprays or washes, argyrol, silver nitrate, and iodoglycerin solution are indicated if the secretions are excessive.

Should severe dyspnea occur from edema, infiltration, or stenosis, appropriate surgical treatment should be undertaken to relieve the symptom. Sequestra of the laryngeal cartilages should be removed with curved laryngeal forceps. Deep abscesses should be evacuated externally or internally according to the indications.

Prolapse of the Laryngeal Ventricles, Eversion of the Ventricles. — **Etiology.** — During syphilitic or tubercular laryngitis a violent attack of coughing may loosen the mucous folds from their attachments to the parts beneath, and allow them to protrude above the larynx or over the true cords (Fig. 172).

Symptoms.—The symptoms are those due to the interference with the respiratory functions, and the movements of the vocal cords.

Treatment.—Painting the parts with astringent solutions will relieve the symptoms insofar as they are due to reactionary inflammation, but will have little influence in overcoming the obstruction caused by the misplaced mucous membrane. The ablation of the dislocated ventricular bands with a snare or laryngeal forceps through the mouth, or after thyrotomy, affords the best means of treatment at our command.

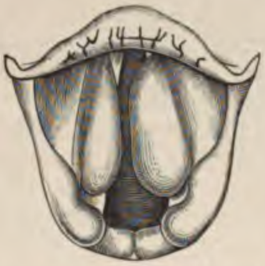

FIG. 172.—Prolapse of the ventricles of the larynx. (Coakley.)

Lupus of the Larynx.—**Pathology.**—Lupus of the larynx is a tubercular infection which is limited to the superficial layers of the mucous membrane. Numerous elevated masses appear on the infected areas and there is a marked reactive proliferation of epithelium. Ulcerations occur sooner or later. The progress of the disease is uncommonly slow, both before and after the ulcerative process is established. Lupus of the larynx, or other portions of the respiratory tract, is almost always *secondary to lupus of the skin*. The process in the respiratory tract begins in the nose and passes downward to the pharynx and larynx. It may, however, be primary in the latter.

Symptoms.—Deglutition is sometimes attended with pain, while the voice is more or less impaired according to the degree and location of the diseased areas. If the true cord is the seat of lupous inflammation, there will be hoarseness or complete aphonia. Dyspnea is present in a few cases in which there is a pronounced tumefaction of the tissues. This rarely occurs, however, as the process is too slowly destructive for the production of edema. Reconstructive infiltration does not figure permanently in this disease, and yet there is but little destruction of tissue, as the process is limited to the superficial layers of the mucosa, and the destructive process is very slow. Pain is usually absent, and any part of the larynx may be affected. Cough may or may not be present.

The general health is usually good.

Laryngoscopic examination reveals a nodular ulcer, with a pinkish-gray bottom, slightly below the surface of the mucous membrane. There is little or no edema, the mucous membrane being slightly hyperemic.

Treatment.—Aqueous solutions of the nitrate of silver, containing from 120 to 480 grains to the ounce, applied locally by means of a laryngeal brush or cotton-wound applicator often exert a very favorable influence on the course of the disease. The topical application of the tincture of iodin, the galvanocautery or fused chromic acid crystals have been used with more or less success. Curettement of the ulcers, followed by the application of a 50 per cent. lactic acid solution, does not seem to act as favorably as it does in true tuberculosis. Excision of the lupous mass, including some of the surrounding healthy tissue, is sometimes followed by improvement.

Leprosy of the Larynx (*Elephantiasis Grecorum; Lepra*).—Leprosy of the larynx is never primary, but is one of the attendant phenomena of general leprosy.

It is characterized by hard nodular infiltration, ulcers, and cicatricial contraction which is apt to produce stenosis. The stenosis is, however, in part due to the infiltration, which, when excessive, involves the lumen of the larynx. Extensive destruction of tissue often occurs, in which the epiglottis is most apt to be involved.

Gouty Inflammation of the Larynx.—This is a rare condition, and is characterized by gouty deposits upon the epiglottis and laryngeal cartilages. These offer a hard and gritty sensation to the sense of touch when probed. The membrane is swollen and yellowish red in circumscribed areas. These symptoms, when taken with the general history of gout, render the *diagnosis* comparatively easy. The *treatment* should be addressed to the relief of the gouty diathesis.

Arthritis Deformans of the Larynx.—Here, as elsewhere, the articular surfaces are the seat of inflammatory deformity. Motion of the vocal apparatus is interfered with, or the ankylosis may be complete.

FOREIGN BODIES IN THE LARYNX, TRACHEA, AND BRONCHI.

Occurrence.—Any solid, fluid, animate or inanimate object may gain entrance into the lower air tract from without by way of the mouth or nose; or from within by the rupture of an abscess of the pharynx, larynx, or other adjacent tissue; and from exfoliation of the laryngeal cartilages.

Symptoms.—The symptoms vary somewhat with the size, location, and physical character of the object.

The *entrance* of the foreign body into the air tract is signalized by a sudden choking, gasping inspiration, and frequently marked dyspnea. There is an anxious expression of the countenance. Suffocation may take place and necessitate an immediate tracheotomy to prevent a fatal termination.

If the foreign body *passes into the lower tract*, the dyspneic symptoms subside or entirely disappear, only to recur at varying intervals. The foreign body may be tolerated for an indefinite length of time, cases being reported in which the foreign body is expelled after having been in the trachea for a lifetime.

After a time inflammatory and ulcerative processes may develop in the parts holding the foreign body. Hoarseness and aphonia, and recurrent attacks of dyspnea mark the later history of many cases of retained foreign bodies. The expectoration may be streaked with blood. A slight febrile movement, closely simulating pulmonary phthisis, may occur in the progress of the reaction inflammation. The foreign body is most apt to enter the right bronchus, as it is in a more direct line with the trachea.

Treatment.—If suffocative symptoms persist, tracheotomy or laryngotomy should be performed. They usually subside, however, before such extreme measures become necessary. The removal of foreign objects from the lower air tract and the esophagus by means of the bronchoscope and esophagoscope has progressed to such a degree at the present time that every suspected case should be given the benefit of the procedure.

Indications for Removal.—Jackson gives the following indications for bronchoscopy.

1. The appearance in the radiograph of a foreign body or of any suspicious shadow.
2. In any case in which there is a clear history of the patient having choked on a foreign body, and in which the foreign body has not been found afterward.
3. In any case in which there are signs of stenosis of the trachea or of a bronchus.
4. In the absence of the history of any foreign body, the patient showing symptoms of pulmonary tuberculosis in which the bacilli cannot be found in the sputum, and especially if the physical signs are at the base (particularly the right base), and above all, if there are physical signs of pleural effusion.
5. All cases of bronchiectasis should be bronchoscoped for foreign bodies and also for local treatment.
6. Every case of dyspnea, except, of course, pneumonia and similar well-understood conditions, calls for bronchoscopy.
7. Every case in which tracheotomy does not relieve, the dyspnea should be bronchoscoped to determine why the tracheal cannula does not give relief.
8. All cases of hemoptysis, which are not definitely proven to be tuberculous, should be bronchoscoped for diagnosis,

and in case of severe bleeding endoscopic packing, as advised by Killian, should be tried.

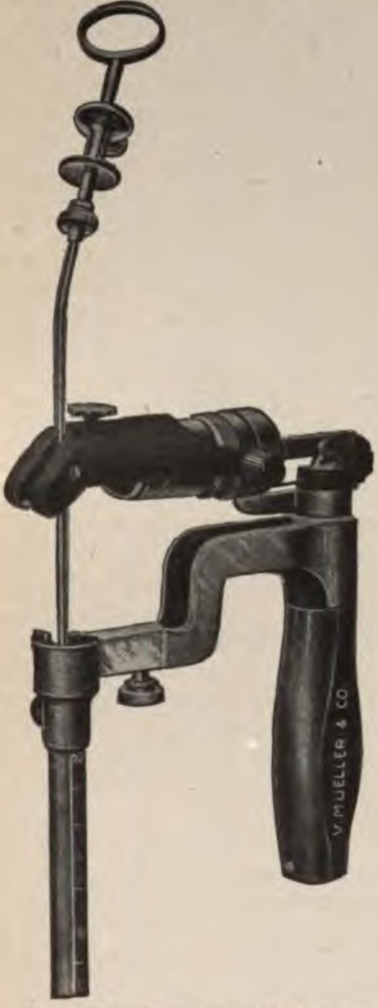

FIG. 173.—Bruening's electroscope for illuminating the laryngeal, bronchial, and esophageal tubes.

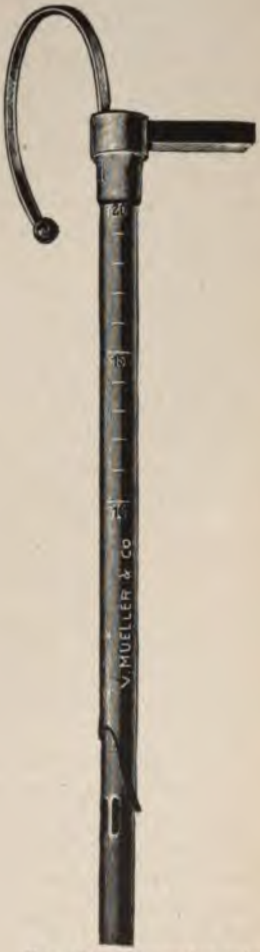

FIG. 174.—Extension tube for bronchioscopic and esophagoscopic work.

9. Every case of paralysis of the recurrent nerve, the cause of which is not positively known.

10. Any case of thoracic disease in which any element of doubt exists, valuable information may be gained by bronchoscopy.

11. In case of doubt, perform a bronchoscopy.

Water hunger is the most serious contra-indication to esophagoscopy.

Anesthetic.—In children under six years of age Jackson recommends that no anesthetic should be used for direct laryngoscopy, peroral bronchoscopy, or esophagoscopy, except that general anesthesia may be advisable in case of very sharp foreign bodies as safety pins or fish hooks threatening perforation from coughing or from vomiting.

Jackson also states that in adults no anesthesia is necessary for esophagoscopy for diagnosis or for foreign bodies, save in case of very large and sharp foreign bodies, to relax the esophageal and accessory musculatures, thereby lessening the liability to local trauma from the extraction of very large foreign bodies that are sharp or rough.

For bronchoscopy for diagnosis, anesthesia of the larynx including the epiglottis is needed.

NEUROSES OF THE LARYNX.

Sensory Neuroses.—**Anesthesia.**—Anesthesia of the larynx may include insensibility to *touch*, *heat*, and *cold*, either separately or together. It may be unilateral or bilateral, and above or below the glottis, according to the seat and nature of the lesion causing it.

Etiology.—It may be due to hysteria, or to a neuritis following diphtheria, syphilis, or meningitis; or to pressure on the trunk of the recurrent laryngeal. It is present in general paralysis of the insane, in tumors at the base of the brain, and in the early stages of tabes dorsalis, or other diseases affecting the medulla.

Treatment.—If the anesthesia is peripheral in origin, the faradic or combined faradic and galvanic currents should be applied locally by means of suitable electrodes, the indifferent electrode being placed on some remote part of the body. Strychnine is a valuable adjunct to the electrical treatment. Iron, phosphorus, and arsenic are indicated in neurasthenics. If due to tumors or other organic diseases of the brain, local treatment is useless.

Hyperesthesia.—Hyperesthesia is a common symptom of acute simple inflammations of the larynx, tubercular laryngitis, and sometimes of chronic laryngitis.

It is present in neurasthenia, anemia, prolonged gastritis and in chronic alcoholism. There may be transitory hyperesthesia of the larynx during teething, menstruation, and pregnancy. It may be the first sign of phthisis, hence often furnishes a valuable aid in the early diagnosis of this important disease.

Increased reflex irritability is the chief *symptom* of laryngeal hyperesthesia. The *prognosis* depends on the cause underlying it. If it is neurasthenia, disordered menstruation, or pregnancy, it is favorable; if due to tuberculosis, it is unfavorable.

The *treatment* should be addressed to the cause, rather than to the hypersensitiveness itself.

Paresthesia.—This is a perverted sensation in the larynx, the patient complaining of a tickling or prickling in the throat, or of the presence of a foreign body. As suggested by the title, the condition is not a real one, but a perverted sensation neurosis. These sensations are to be accounted for by obstructed venous circulation and hypertrophy or other diseased states of the lingual and faucial tonsils. In other cases they are due to slight tissue changes, as in incipient tuberculosis.

Neuralgia.—The usual signs of neuralgia are present, as pain and tender spots, the pain being either constant or paroxysmal. It may be spontaneous or caused by attempted phonation, as in hysterical individuals. Phosphorus, arsenic, quinine, iron, and strychnine should be administered internally, and the galvanic current applied locally. The positive electrode of the battery should be applied through the mouth to the laryngeal mucosa, while the negative pole should be placed on some remote part of the body. Should the poles of the battery be reversed in their application, the pain will be increased rather than decreased. The dose of the current should be limited to about 5 ma., as more than this is apt to excite inflammatory reaction, or produce actual destruction of tissue by electrolysis.

Hyperkinetic Neuroses (*Spasmodic Neuroses of the Larynx*).—The spasms may be either *tonic* or *clonic*.

Tonic Spasms of the Laryngeal Muscles.—Their **origin** may be (*a*) *central;* (*b*) from *irritation* of the *trunk* of the *recurrent laryngeal;* and (*c*) from *reflex irritation.*

Tonic Spasms of Central Origin.—In tabes dorsalis, spasm of the adductors of the larynx occurs. *Clinical picture:* Sudden dyspnea with loud inspirations, the cords remaining in adduction for some time. It also occurs in tetanus, tetany, and hydrophobia.

Tonic Spasm from Irritation to the Trunk of the Recurrent Laryngeal.—When the injury is transient and slight, there will be a laryngeal spasm which is a forerunner of paralysis. Aneurism of the arch of the aorta, cancer of the esophagus, pleuritic adhesion of the right apex of the lung, and tumors of the mediastinal glands may cause the irritation. Slight lesion may also occur in tabes.

Tonic Spasms from Reflex Irritation.—These may occur from irritation to the larynx, fauces, and neighboring parts. In highly sensitive children, irritation in a remote part of the body may cause adduction spasms. The latter condition has been described as *laryngospasmus infantum*, and is usually due to intestinal irritation, tapeworms, a tight prepuce, constipation, or adenoids.

Clonic Spasms of the Laryngeal Muscles.—These are always of *central origin.*

They consist of rhythmical inward movements of the cords. The condition may last but a few moments, or may persist for many months. The pillars of the fauces are also often affected in a like manner.

The *tonic* and *clonic* spasms may be present in the same case, especially in the depressors of the epiglottis. The diseases most often *causing* clonic spasm of the larynx are syphilis, meningitis, and intracranial tumors.

Treatment of Laryngeal Spasms.—*Of Central Origin.*—It should be *symptomatic*, as the diseases causing it are usually incurable. Prolonged *tonic* spasms of the adductors, resulting in marked dyspnea, should be relieved by tracheotomy. Morphine injections will reduce excessive excitability.

Of Reflex Spasms.—These are usually seen in children, and should be arrested, as more or less danger attends them. The child should be placed in a warm bath and the posterior part of the chest douched with cold water. The intestinal tract, portal circulation, and genitalia should receive careful attention and, if necessary, appropriate treatment.

Of Spasms from Irritation of the Recurrent.—Little can be done for spasms from irritation of the recurrent laryngeal nerve, as it is usually due to intrathoracic pressure from some grave tumor or other morbid condition.

Laryngeal Vertigo (*Laryngeal Apoplexy; Laryngeal Syncope; and Complete Glottic Spasm in Adult*).—It is characterized by transient irritation in the throat, followed by a fit of coughing, dimness of vision, dizziness and unconsciousness, the patient falling to the floor.

Symptoms.—The face is flushed. There may be epileptiform convulsions, which, after a little while, are followed by a return of consciousness. After such an attack all signs of the condition disappear. The seizure is clinically much like apoplexy with laryngeal aura and laryngeal spasm, which is continued long enough to produce unconsciousness. Such spasms are liable to occur in neurasthenics and in tabes. Other signs and suggestive family history which characterize epilepsy, tabes, and neurasthenia should be sought for before pronouncing the case one of laryngeal vertigo.

Treatment.—The treatment should be addressed to the correction of alimentary and portal disorders; and to the excretory organs of the body. Tonics, arsenic, and phosphorus are sometimes of value in preventing a return of the laryngeal disorder.

Nervous Cough.—*Definition.*—This is a spasmodic, croupy, or even musical, laryngeal cough, for which no physical cause can be assigned.

Symptoms.—Persons thus affected are neuropaths, presenting other stigmata of neurosis. The cough is usually persistent during the daytime, being increased upon excitement, and subsiding during sleep. Upon awakening in the morning it returns with renewed vigor.

Treatment.—Ten-grain doses of the bromid of potassium or sodium should be given internally three times a day. The larynx should be sprayed with aromatic oily solutions of menthol and eucalyptol. Systemic tonics, laxatives, outdoor exercise, together with occupancy of a room with abundant exposure to sunlight and fresh air should be made a part of the daily régime.

Mogiphonia.—This is characterized by difficulty in maintaining the tension of the vocal cords while singing or during forced, accentuated speaking. In ordinary conversation no difficulty is experienced.

Rest, massage, friction, and regulation of alimentary and portal disturbances afford the best means of treatment.

Chorea of the Larynx (*Laryngeal Nystagmus*).—This condition is quite like chorea in other parts of the body, and is characterized by frequent recurrences of a sharp, dry, noisy cough resembling a bark or yelp of a dog. Here, as in chorea of the arms and face, the disease is usually manifested in females about the age of puberty.

Symptoms.—The yelp or bark consists of a sudden expiratory effort, or of a series of similar noises gradually diminishing in intensity. Choreic movements are usually, though they may not be, present in other parts of the body. The voice is unaffected, but may betray a slight jerkiness in articulation.

Treatment.—The patient should be treated as for general chorea, with the addition of sedative inhalations of a hot vapor of the compound tincture of benzoin; or of the vapor from a hot infusion of hops or hyoscyamus. Bromids and arsenic should be administered internally, the bowels and portal circulation regulated, and the activity of the skin established by friction and cold douches.

Hypokenitic Neuroses (*Laryngeal Paresis and Paralysis*).—*Motor innervation* of the laryngeal muscles is by the superior and recurrent laryngeal nerves. The *recurrent* is most frequently involved.

Total Unilateral Paralysis of the Recurrent.—The clinical picture is as follows:

(*a*) The affected cord is fixed midway between the respiratory and phonatory positions—*i. e.*, in the cadaveric position. Neither the abductors nor the adductors are acting. The cord is in a state of passive equilibration.

(*b*) The free edge of the affected cord is more concave than normal.

(*c*) The arytenoid cartilage on the affected side is rotated somewhat inward, thus allowing the vocal process to project into the chink of the glottis, and convert the concave edge of the cord into two concave segments.

(*d*) The arytenoid cartilage on the unaffected side moves forward during phonation somewhat in advance of the one on the affected side.

(*e*) During phonation the paralyzed cord and the arytenoid cartilage remain fixed; while those upon the unaffected side move inward somewhat beyond the median line. The exaggerated inward movement is a compensatory effort at vocalization.

(*f*) The glottic opening is oblique from before backward toward the affected side.

(*g*) The arytenoid muscle on the affected side sometimes twitches, as it is occasionally supplied by the superior laryngeal, which is not usually involved. This sign should enable the observer to exclude ankylosis and prove the nervous origin of the impairment.

(*h*) The epiglottis is usually innervated by the superior laryngeal, but is occasionally supplied by the recurrent. When by the latter, the epiglottis may be seen to twitch upon the sound side.

(*i*) The voice is at first feeble, slightly roughened, but not hoarse. Later on these conditions disappear and the voice becomes nearly natural. This is due to compensatory function on the unaffected side.

(*j*) There is excessive waste of air during phonation, and the respiratory act is necessary after speaking a few words.

Total Bilateral Paralysis of the Recurrent.—The clinical picture is as follows:

(*a*) There is complete aphonia, as both cords are in the cadaveric position.

(*b*) There is great waste of air upon attempted phonation.

(*c*) There is no motion of the cords, as both the adductors and abductors are paralyzed.

Paralysis of the Abductors.—In this condition a portion of the fibers of the recurrent pharyngeal are involved, and the following *clinical picture* is presented:

(*a*) There is almost complete closure of the glottis, giving rise to

(*b*) Dyspnea; and

(*c*) Stridor. Both are increased during sleep or upon slight irritation of the larynx.

(*d*) The dyspnea and stridor are greater during inspiration.

(*e*) The voice is usually unaffected in ordinary conversation.

(*f*) The cords remain almost parallel, being more nearly approximated during inspiration. This sometimes results in profound dyspnea.

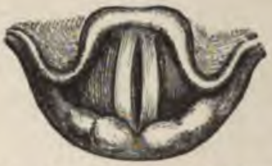

FIG. 175.—Position of the vocal cords in respiration in bilateral abductor paralysis. (Cohen.)

Prognosis of Recurrent Laryngeal Paralysis.—In paralysis either of the muscles of abduction, adduction, or both combined, the prognosis is usually serious, as the conditions causing it are incurable or very grave. The dyspnea may be so marked as to call for tracheotomy.

Laryngeal Paralysis from Various Causes.—It may result from injury to the pneumogastric by fracture at the base of the skull; or to the spinal accessory; or from bulbar disease.

Postdiphtheritic bulbar degeneration sometimes suddenly develops and causes laryngeal paralysis. When disseminated sclerosis invades the medulla and involves the laryngeal centres, the symptoms are bilateral. Tabes is the type of degeneration which most often impairs the laryngeal centres. In this disease the paralysis may be bilateral or unilateral, complete or incomplete.

Atrophy of the Vocal Cords.—Atrophy may arise from any condition which prevents the use of the vocal organs, as paralysis of central or peripheral origin; and from inflammatory infiltration and degeneration of the muscular fibers themselves. It may also follow ankylosis of the arytenoid cartilages, or prolonged fixation of the cartilages from rheumatic laryngitis. Faradism may be applied locally in those cases which have a prospect of ultimate recovery.

Hysterical Paralysis (*Functional Aphonia*).—*Etiology.*—Great mental or physical exhaustion resulting from sickness, prolonged anxiety, or physical exertion is sometimes attended by complete loss of the voice. It may be due to the inhibition of the will centres brought about by fright or powerful suggestion. Occasionally chronic laryngitis seems to be the cause.

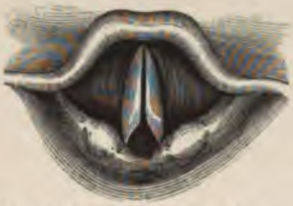

FIG. 176.—Paralysis of the arytenoideus. (Coakley.)

Symptoms.—The voice (voluntary muscular action) is lost, while coughing and laughing are phonetic, as they are involuntary acts. Speech, being voluntary, is inhibited. *Inspection* shows the cords separated, moving slightly inward upon attempted phonation, and slightly outward upon deep inspiration (Fig. 176). The arytenoideus muscle may alone be affected and cause hoarseness and fatigue upon phonation.

Treatment.—Tonics, nutritious food, plenty of exercise in the open air, sunshine, freedom from mental anxiety, excessive physical exercise will do much toward effecting a cure. Suggestive therapeutics will aid greatly in overcoming the inhibition of the will, one of the most important etiologic factors of the condition.

Asthma. — Nasal Disease as an Etiologic Factor. — One of the common sources of reflex irritation in the production of asthma is the nose and throat. Probably the most important reflex is from the nasal mucous membrane. Such conditions as polypoid or suppurative ethmoiditis, hypertrophic turbinate, deviated septum in contact with a portion of the outer nasal wall, diseased tonsils, etc., may cause asthma by reflex irritation of the vagus.

Treatment.—The treatment of asthma of nasal origin consists in the correction of the nasal lesion.

TUMORS OF THE LARYNX.

Benign Tumors.—Classification.—Papillomata, fibromata, cystomata, myxomata, adenomata, lipomata, angiomata, and enchondromata.

Etiology.—Hyperemia is regarded by some as a cause, while other observers of equal repute do not so regard it. The same is true of catarrhal inflammations.

Benign tumors are most common in middle life, but are occasionally found in the very young as well as in the old. They are most common in males, probably on account of a more exposed life and pernicious habits. Overtaxing the voice, as in violent singing, street crying, auctioneering, and public speaking, seems to be a prominent cause. The inhalation of irritating vapors, has more or less influence in their causation. Nasal obstruction probably exerts some influence.

Symptoms.—The voice is impaired in about nine-tenths of the cases, the impairment usually being in the form of hoarseness or aphonia. Occasionally, however, it may be double—*i. e.*, gives off two tones at once. This is caused by small growths which divide the cords into two segments, each segment giving forth a different tone. If there is no hoarseness or other impairment of the voice, its volume and strength are diminished. In about 15 per cent. of the cases there is marked dyspnea. Cough may or may not be present. The expectoration is scanty and sometimes streaked with blood. Deglutition is interfered with when the growth is upon the epiglottis, in the hyoid fossa, or upon the wall of the pharynx. There may be some discomfort, but no pain. In adults the mucosa is not usually inflamed. In children, however, the growths are usually attended by laryngitis, which gives rise to nocturnal dyspnea.

The *laryngoscopic image* varies with the location and character of the growth.

The Cough-spots of Stoerck.—The sensitive areas known as "the cough-spots of Stoerck" are located on the (*a*) posterior commissure between the arytenoid cartilages; upon the (*b*) posterior wall of the larynx and (*c*) trachea; and upon the (*d*) bifurcation of the trachea.

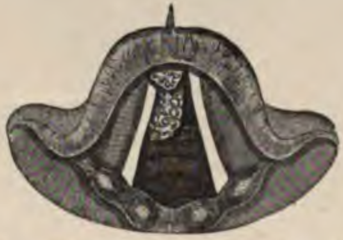

FIG. 177.—A small papilloma springing from the anterior commissure and resting on the right vocal cord. A papilloma is shown arising in the subglottic region of the larynx beneath the right vocal cord. (Cohen.)

Diagnosis.—*Papillomata.*—They are soft, pinkish-gray or whitish-gray, wart-like growths (Fig. 177). They are usually movable, and grow from the anterior angle of the cords. The author observed a case in which they grew from the whole length of the cords and from the under surface of the epiglottis.

Fibromata.—They are firm to the touch, have a rounded nodular contour (Fig. 178), and the mucous membrane covering them is somewhat injected. They are either sessile or imbedded in the surrounding tissues. They most often grow from the anterior portion of the cords. In general appearance they somewhat resemble cystomata and chondromata. Chondromata never grow from the cords, and are much firmer to the touch; while cystomata are soft, movable, and semitranslucent.

Cystomata.—They pit upon probe-pressure and regain their rotundity at once. They are small, roundish, pinkish-gray, semitranslucent tumors. When lying across the chink of the glottis, they appear almost transparent.

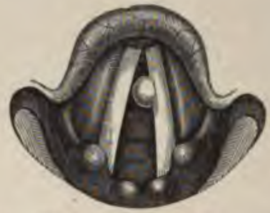

FIG. 178.—Fibroma on the left vocal cord.

Chondromata.—As chondromata grow from the cartilaginous box, they are covered with healthy mucous membrane. They develop very slowly, the cricoid cartilage being most often affected. In this location they may resemble perichondritis or carcinoma. In *perichondritis* there is a history of sudden onset, followed by marked dyspnea, which renders the differentiation clear. *Carcinoma* of the larynx rarely grows below the glottis, hence need not be confounded with chondroma of the cricoid.

Laryngeal Polypus.—This is a rounded semitranslucent tumor usually attached to the vocal cords, as shown in Fig. 179. They are slightly pedunculated and more dense than polypi of the nasal mucous membrane.

Treatment.—This is chiefly *surgical.* The direct or indirect method of operating may be used.

Direct Method.—This is done by means of the Killian suspension instrument. This method is probably the most accurate inasmuch as the operator has both hands free and a view of the larynx is continually present. Lynch has devised a number of straight, slender instruments which greatly facilitate the direct method of operating.

Indirect Method.—Nearly all benign tumors may be removed through the mouth with a snare, cautery, or one of the curved curettes. If the growth is pedunculated, the snare loop may be insinuated around it; and if it is sessile, one of the laryngeal biting or cutting curettes should be used. Small growths upon the edge of the cords are best removed with the serrated laryngeal forceps. Care should be exercised to avoid injury to the cords, as permanent impairment of the voice might follow.

Local anesthesia should be produced by the application of a 20 per cent. solution of cocain to the laryngeal mucous membrane, otherwise the spasmodic movements of the pharynx and larynx will interfere with the performance of this operation. Great dexterity from large experience is required for these operations. The operator does not see the growth, but an inverted image of it. The same is true of the cutting end of the laryngeal instrument. He is guided by the reverse movements observed in the laryngeal mirror. This makes the operation a very difficult one. The beginner should practise laryngoscopic examination and instrumentation upon a large number of persons before attempting to remove morbid tissue from the larynx.

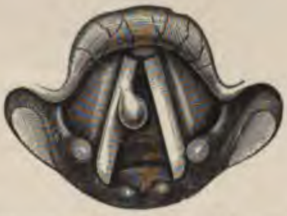

FIG. 179.—A polyp on the right vocal cord.

Malignant Tumors.—**Sarcoma.**—*Definition.*—Sarcoma of the larynx is an embryonic connective-tissue growth usually occurring between the twentieth and fortieth years. The larger celled tumors grow slowly; while the smaller celled ones develop rapidly. The tumor is rarely found in the larynx, being in the proportion of one to sixty-two of carcinoma.

Symptoms.—Vocalization and respiration are impaired early. There is a spasmodic, hacking cough, due to the presence of secretion. Cough is a distressing symptom when the growth is infraglottic. Dysphagia is present when it extends upward, involving the epiglottis and contiguous tissues. Erosion and ulceration appear early, but pain is rarely present. The sputum is tinged with blood, although hemorrhage is not so common as in sarcoma of the nose. The tumors are usually small in size when recognized, as they interfere with respiration before becoming large. The growth does not extend to the adjacent tissues, but if it begins in them it often extends into the larynx. The author recently saw a case in which the posterior wall of the pharynx and larynx were involved, the growth being about the size of a large filbert. A cachexia sometimes develops, and is due to obstructed respiration and deglutition.

Diagnosis.—The tumor is soft and grumous, with an irregularly rounded outline, and is of a pinkish-gray or purplish-gray color. A portion should be removed and submitted to microscopic examination.

Prognosis.—The prognosis is much more grave than that of sarcoma of the nose. It is almost as fatal as carcinoma of the larynx. The average length of life is less than two years.

Treatment.—Extirpation is sometimes followed by a cure. Laryngo-fissure or a complete resection or extirpation of the larynx is usually indicated, as a less extensive operation will be of but temporary value. A preliminary tracheotomy should be performed before removing the larynx, as the anesthetic must be administered through the artificial opening.

If laryngo-fissure is done, it is the author's custom to use a local anesthetic. The procedure is not an uncomfortable one, and the dangers attending the operation are greatly lessened.

Carcinoma.—*Etiology.*—Heredity exerts an important influence in a small percentage of the cases. It is more frequent in males, and occurs late in life.

Symptoms.—These will depend upon the location, size, and stage of development of the cancer. If it is located upon the cords or ventricular bands, the voice will be affected. If the posterior commissure is affected, there will be cough; while if located on the anterior wall, the function of the epiglottis will be impaired, thus giving rise to dysphagia. If it is in the ulcerative stage, there will be a foul odor attended by cough, while pain will be more pronounced.

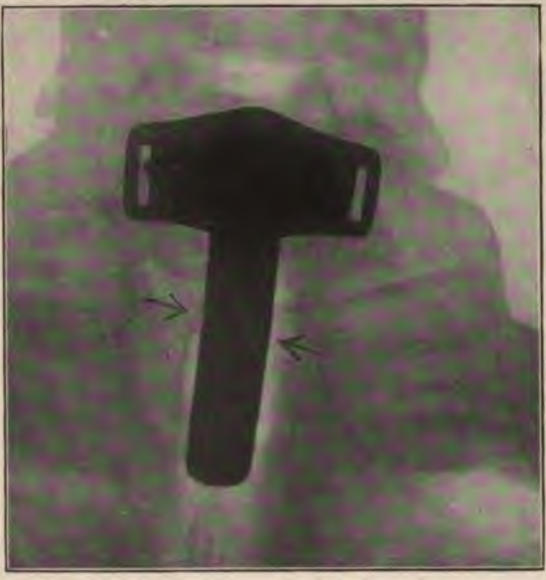

FIG. 180.—Showing upper limit of trachea after laryngectomy for carcinoma.

At first the voice is weakened and changed in quality or pitch; after a time it is entirely lost. Hemorrhage may occur after the ulceration has formed. It is not usually a fatal or dangerous complication. The cancerous cachexia may or may not be present in the early and middle stages of the disease.

Diagnosis.—This must be based upon the location of the tumor, the character and extent of the ulceration, the existence of pain after a somewhat delayed period, and upon a microscopic examination of a piece of the diseased tissue. This must be removed from a non-ulcerated portion of the growth, as the granular surface of the ulcer will not show characteristic *inward proliferation of the epithelium.* The growth is nearly always supraglottic.

Prognosis.—Intrinsic cancer of the larynx, in the majority of cases, begins as a growth on one of the vocal cords, and can therefore be completely removed by laryngo-fissure. If the growth has extended to the anterior commissure, the prognosis is not so good, as the growth is cut through when the thyrotomy is done, and a recurrence frequently takes place in the line of incision.

Treatment.—Surgical treatment gives very good results, especially when compared with those obtained by surgical treatment of cancer in other parts of the body.

The operation of choice, when the cancer is limited to the cords, is the laryngo-fissure. Schmiegelow reports 66 cases, in 33 of the cases the primary seat of the growth was in the vocal cords, 18 cases had no special localization, 5 were in the ventricular band, 4 in the arytenoid region, 2 in the sinus (Morgagni), and one in the epiglottis. Of the 33 cases where the primary seat was on the vocal cords and were operated by thyrotomy, 18 were cured and 15 died or relapsed.

Total or hemilaryngectomy should be resorted to when the growth has invaded to any extent the structures other than the cord.

Palliative remedies should be used locally to mitigate the distressing symptoms. The secretion can be removed by gargles containing 10 per cent. of carbonat of sodium. Powders containing morphine or cocain, or both combined, afford relief to the sharp, lancinating pains. The fetor may be removed or modified by solutions containing 2 per cent. of permanganat of potassium or an equal amount of carbolic acid.

PRESCRIPTIONS FOR EAR, NOSE, AND THROAT.

The following prescriptions have been selected from textbooks and from those used by the author. Some of them have appeared in the text, but most of them are additional remedies which have been found to be valuable. They are classified under the diseases in which they are most useful.

Simple Cleansing Solutions:

℞—Sod. bicarb.,	
Sod. boratis, sod. chloridi	āā ℥j—M.

Sig.—Add a teaspoonful to a pint of lukewarm water and use with a syringe or atomizer to cleanse the nose from thick mucus and crusts.

℞—Sod. bicarb.	gr. xv
Sod. bibor.	gr. xv
Acid. carbol.	gr. iv
Glycerin.	♏xlv
Aquæ	ad ℥j—M.

Sig.—Dobell's solution. Dilute with equal parts of water and use with atomizer; or snuff up the nose.

℞—Acidi acetici	ʒiiss
Glycerin.	ʒiij
Aquæ	ad ℥x—M.

Sig.—To be used as an antiseptic and stimulating wash in the nose and nasopharynx in the course of the exanthematous fevers.

Hay Fever, or Hyperesthetic Rhinitis:

℞—Acidi carbolici	gr. xxx
Ammonii carbonatis	℥j
Pulv. carbonis ligni	℥j
Olei lavandulæ	♏xx
Tinct. benzoin. co.	℥ss—M.

Sig.—Uncork and inhale to relieve the congested condition of the mucous membrane in acute coryza and hay fever.

℞—Acidi carbolici	gr. xxx
Ext. pini canadensis dest.	♏xx
Liq. vaselin.	℥j—M.

Sig.—To be used in the acute stage of hay fever for the anesthetic effect of the carbolic acid. (Ingals.)

℞—Zinci valerianatis	gr. j
Pil. asafœtid. co.	gr. ij—M.

Ft. pil. j.

Sig.—Give one or two pills two or three times a day to relieve the neurasthenic symptoms occurring in hay fever. (Sir Morell Mackenzie.)

℞—Glycerit. acid. carbol. ℥j
Quininæ hydrochlor. ʒj—M.

Sig.—To be dissolved with the aid of heat and one two-thousandths part of perchlorid of mercury added. To be applied with caution to the nasal mucous membrane every two or three days for the relief of the acute symptoms of hay fever. (Sir Andrew Clark.)

℞—Acid. chromici cryst. gr. ⅛, ⅙, ½
Aquæ dest. ℥j—M.
Ft. nebulæ and use to check sneezing.

Chronic Rhinitis:

℞—Potass. chlor. gr. x–xx
Aquæ ℥j—M.
Sig.—To be used as a spray in rhinitis with collapse.

℞—Eucalyptol ♏xv
Menthol gr. xv
Camphor gr. lx
Ol. pini compilonis ʒss
Ol. rosæ ♏ij
Liq. vaselin q. s. ad ℥ij—M.

Sig.—Use in the nose with an atomizer in mild cases of hypertrophic rhinitis.

℞—Ichthyol gr. xlviij
Lanolin,
Vaselin āā ʒiv—M.

Sig.—Use on a cotton-wound applicator to massage the nasal mucous membrane in chronic forms of rhinitis.

Ozena and Atrophic Rhinitis:

℞—Hydrarg. ammoniati gr. iv
Pulv. sacch. alb. ℥ss—M.

Sig.—Insufflate into the nose to stimulate the mucous membrane in ozena.

℞—Hydrarg. oxidi rubr. gr. iv
Pulv. sacch. alb. ℥ss—M.
Sig.—To be blown into the nose after cleansing in ozena.

℞—Iodoformi gr. v–x
Menthol gr. iij–v
Lanolin ℥ss
Liq. vaselin ℥ss—M.
Ft. ung.

Sig.—To be applied to the interior of the nose with a brush in ozena or ulcer of the septum.

℞—Creolin ♏j–iv
Aquæ ℥j—M.

Sig.—Antiseptic and deodorant, to be used in atrophic rhinitis, syphilitic and other ulcerations, and in diseases of the accessory sinuses.

℞—Sanguinariæ canadens. . . . . ♏v–xxx
Aquæ tepid . . . . Oss—M.
Ft. lotio.

Sig.—To be used with an atomizer or syringe night and morning for ozena.

℞—Zinc sozoiodol . . . . gr. xlviij
Talc. . . . . q. s. ℥j—M.

Sig.—To be blown into the nose in atrophic rhinitis after cleansing the nose from crusts.

Diphtheria and Pseudodiphtheria:

℞—Menthol . . . . gr. xlviij
Toluol . . . . ʒij
Sol. of perchlorid of iron (fort.) . . . . gr. xx
Alcohol absol. . . . . q. s. ad ℥j—M.

Sig.—To be applied locally in diphtheria or pseudodiphtheria.
(Löffler.)

℞—Acidi lactici . . . . ʒj–ij
Aquæ . . . . ℥j—M.

Sig.—To be used with an atomizer in diphtheria as a solvent of the membranous exudate. (L. Browne.)

℞—Acidi sulphurosi . . . . ℥ss–j
Aquæ . . . . ad ℥x—M.

Sig.—To be used as a gargle in diphtheria, pseudodiphtheria, and in mycosis.

Empyema of the Accessory Sinuses:

℞—Aristol.
℞—Europhen.
℞—Nosophen.
℞—Iodol.
℞—Iodoform.

Sig.—To be blown into the nares in empyema of the accessory sinuses.

Relaxed Uvula:

℞—Aluminis,
Acidi tannici . . . . āā gr. lx
Aquæ rosæ . . . . ℥x—M.

Sig.—To be used as a gargle in relaxation of the uvula and congestion of the fauces.

Hemostatics:

℞—Antipyrin . . . . gr. xv
Aquæ . . . . ad ℥j—M.

Sig.—To be used in the nose and throat as a hemostatic.

℞—Pulv. fol. matico,
Pulv. amyl. exsic. . . . . āā equal parts—M.

Sig.—To be insufflated into the nose for epistaxis.

Adenoids:

℞—Glycerit. acid. tannici		ʒiij
Aquæ	ad	℥iij—M.

Sig.—One teaspoonful to a wineglassful of warm water, to be injected down the nose night and morning, as a remedy for postnasal adenoids.

Dry Catarrh of the Nose, Pharynx, and Larynx:

℞—Iodini		gr. v–xx
Potassii iodidi		gr. x–xxx
Ol. gaulther. (ol. menth. pip.)		♏v
Glycerin.		℥j—M.

Make four solutions, varying the amount of iodin and potassium iodid between the limits given in the prescription.

Sig.—In dry rhinitis, pharyngitis, and laryngitis begin with the weakest solution and gradually increase to the strongest to stimulate the glandular function of the mucous membrane.

Acute Tonsillitis and Pharyngitis:

℞—Guaiacol,		
Olive oil	āā	ʒiv—M.

Sig.—Apply to the fauces of pharynx in acute tonsillitis and pharyngitis.

℞—Argent. nitrat.,		
Aquæ dest.	āā	ʒiv—M.

Sig.—It may be used instead of the guaiacol mixture.

One or two applications of the above mixture are often sufficient to arrest acute inflammations of the throat and pharynx if applied in the first stage.

℞—Red gum lozenges (Wyeth).

Sig.—To be dissolved in the mouth at frequent intervals in mild attacks of acute pharyngitis.

℞—Guaiacol		℥ss
Ol. amygdalæ dulcis		℥ss—M.

Sig.—To be applied with a cotton-wound probe in acute inflammations of the throat.

It is also applied to relieve pain.

Laryngitis:

℞—Tr. iod.		ʒj
Glycerin.		℥ss
Aquæ	ad	℥iij—M.

Sig.—To be used to moisten compress in laryngitis.

℞—Ammonii carbonatis		gr. ij–iv
Tr. scillæ		♏x
Tr. digitalis		♏v
Liq. strychninæ		♏ij
Infus. cascarillæ	ad	℥j—M.

Sig.—Give one teaspoonful every three to six hours as an expectorant in subacute catarrh of the larynx.

℞—Eucalyptol		♏x
Menthol		gr. v
Camphor		gr. v
Liq. vaselin	q. s. ad	℥j—M.

Sig.—To be used in acute laryngitis after cleansing the throat with aqueous solutions.

Tubercular Laryngitis:

℞—Menthol		gr. xlviij
Ol. oliv.	q. s.	℥j—M.

Sig.—To be applied to the larynx in laryngeal tuberculosis with a cotton-wound applicator or an atomizer. (Rosenberg.)

℞—Ol. eucalyptol		ʒij
Ol. terebinth.		ʒj
Magnesiæ carb. levis		ʒij
Aquæ	q. s. ad	℥iij—M.

Sig.—A teaspoonful in a pint of hot water. Inhale the vapor to loosen the secretion and allay the cough in tubercular laryngitis.

Nervous Dyspepsia of Singers:

℞—Quininæ sulphatis, Acidi carbolici, Extract. krameriæ	āā	gr. ss
Pep.		gr. ij—M.

Ft. pilula.

Sig.—Take one before each meal at which meat is taken in cases where there is sluggish digestion with flatulency.

Syphilis:

℞—Potassii chloratis		gr. xc–cxx
Glycerin.		ʒij
Aquæ	ad	℥x—M.

Sig.—To be used as a mouth wash in syphilis during the administration of mercury.

℞—Liq. hydrarg. nitratis		ʒj-iv
Aquæ	q. s. ad	℥j—M.

Sig.—To be applied to the sloughing ulcer of tertiary syphilis. As the application is very painful, cocaine should first be applied.

Inspissated Cerumen:

℞—Sodii bicarb.		gr. x
Glycerin.		ʒj
Aquæ	ad	ʒiv—M.

Sig.—Drop into ear three times daily to soften inspissated cerumen preparatory to removing with a syringe.

℞—Hydrozone (15 vol.), Aquæ	āā	ʒiij—M.

Sig.—Same as preceding.

Eczema of the Auricle and Meatus:

℞—Acidi carbolici		gr. v
Spirit. rectif.		ʒij
Glycerin.		ʒij
Aquæ		ʒiv—M.

Sig.—Paint external meatus in scaly eczema with pruritus.

℞—Liniment. calcis,		
Ung. hydr. nitratis	āā	ʒiss
Liq. carbonis detergent.		♏xij
Ung. zinci oxid.	ad	℥j—M.

Sig.—For external use in scaly eczema of the auricle or external auditory canal.

℞—Liquor plumbi subacet.		ʒj
Glycerin.		ʒij
Aquæ	ad	℥iv—M.

Sig.—In eczema or diffused inflammation of the auricle and meatus the parts should be frequently sponged with the lotion.

Furunculosis of the External Meatus:

℞—Menthol		gr. ij
Iodoform		gr. iv
Lanolin		ʒij
Vaselin		ʒij—M.

Sig.—Apply on cotton plug in furunculosis of the external meatus.

℞—Acidi carbolici		gr. xx
Glycerin.,		
Aquæ	āā	ʒiv—M.

Sig.—Local application to relieve pruritus.

Mastoid Periostitis:

℞—Tinct. iodi.

Sig.—In mild cases of mastoid periostitis and in disease of the labyrinth; external use.

Anodyne Mixtures:

℞—Bismuthi subnit.,		
Pulv. acaciæ	āā	ʒij
Iodoformi		ʒss
Morph. sulph.		gr. xx
Acidi tannici		ʒss—M.

Sig.—Insufflate into the larynx half an hour before taking food in cases of painful deglutition in tuberculosis and malignant disease.

The iodoform may be omitted if the odor is disagreeable to the patient.

℞—Cocain		gr. xx
Iodoformi		ʒij
Zinc. stearat.		ʒj—M.

Sig.—Blow into the throat or nose for the relief of pain in tubercular or malignant ulcers.

℞—Camphoræ (reduced to fine powder with a few drops of rectified spirits),		
Chloral hydrat	āā	℥ss—M.

Sig.—To be applied externally as an anesthetic in neuralgic and other painful affections of the throat.

℞—Acidi carbolici (1 to 5 per cent.),		
Glycerin.	āā	℥j—M.

Sig.—Drop into ear to relieve pain in middle-ear inflammation.

Suppurative Otitis Media:

℞—Acidi carbolici		gr. xx
Sp. vini rectif.,		
Aquæ	āā	℥j—M.

Sig.—Instil into ear after drying and cleansing in chronic purulent inflammation of the middle ear.

℞—Iodoformi	ʒss
Sp. vini rectif.	℥j—M.

Sig.—Instil in chronic purulent inflammation of the middle ear.

Tinnitus Aurium:

℞—Infus. digitalis	℥iv—M.

Sig.—Two teaspoonfuls twice daily in pulsating forms of tinnitus.

℞—Sodii brom.	gr. xv–xxx

Sig.—Dissolve in a tumbler of water and take at bedtime; in cerebral symptoms and vertigo occurring in the course of ear disease.

℞—Ammonii chloridi		gr. xx
Sodii iodidi		gr. iij
Ext. glycyrrh. liq.		♏xx
Aquæ	ad	℥j—M.

Sig.—To be used internally in chronic nasopharyngitis associated with middle-ear inflammation and tinnitus. Preferably given at 11 A.M. and 4 P.M.

℞—Potassii iodidi	gr. xxx
Camphoræ	gr. vj
Ol. menth. pip.	♏v
Ung. simpl.	ʒiv—M.

Sig.—To be applied with friction to the mastoid surface for the relief of tinnitus aurium. (Gruber.)

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